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A. W. Hoisholt
Stockton State Hospital
Nov. '98.

ATLAS AND ESSENTIALS
OF
PATHOLOGICAL ANATOMY

BY

DR. O. BOLLINGER

OBERMEDICINALRAT AND PROFESSOR

VOLUME I.

CIRCULATORY, RESPIRATORY, AND DIGESTIVE APPARATUS, INCLUDING THE LIVER, BILE DUCTS, AND PANCREAS

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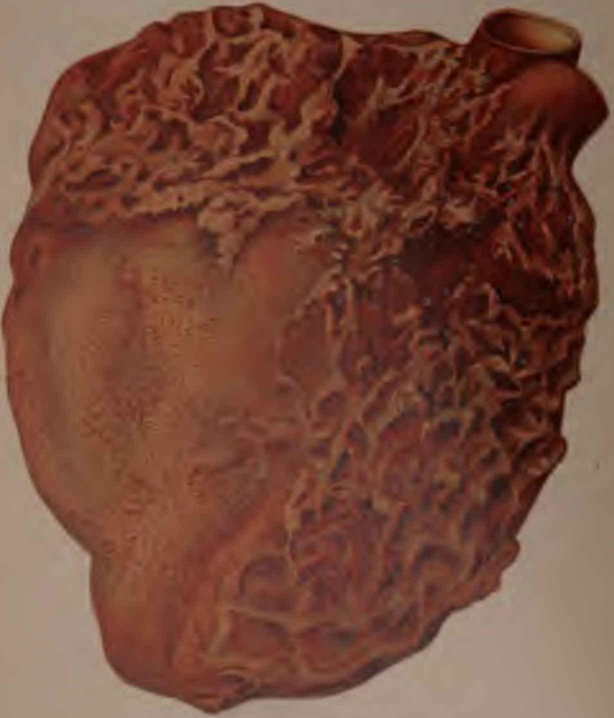


Explanation of Plate 1.

ACUTE SEROFIBRINOUS AND HEMORRHAGIC PERICARDITIS.

(The heart is not opened.)

The greater portion of the pericardium is covered with a reddish-gray pseudo-membrane, whose surface presents at many points a reticulated and villous arrangement. On the anterior surface of the right ventricle toward the apex the inflamed epicardium appears smoother, with a reddish-yellow discoloration; at this point the exudation shows beginning organization and can be removed only with difficulty. The interior of the pericardium contains at the same time about a wine-glassful of a turbid sero-sanguinolent fluid mixed with a few fibrinous flakes.

In the present case the pericarditis was obviously due to a preceding tuberculous pneumonia and pleuritis. Other associated conditions found were long-standing cheesy tuberculosis of the peribronchial and mesenteric lymphatic glands, and acute miliary tuberculosis of the liver, spleen, and kidneys.

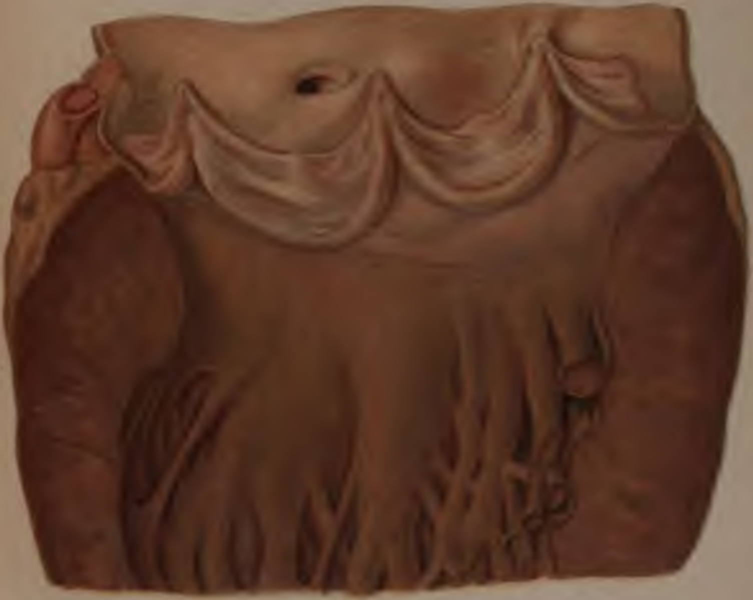
Explanation of Plate 2a.

FATTY DEGENERATION OF THE HEART MUSCLE.

(Degenerative Fatty Heart.)

The subendocardial layers of the muscle are seen to be diffusely discolored, the tint being pale yellow and like a tiger skin. At the same time the muscle is very friable, or of almost buttery consistence, easily disintegrated with the finger. As a rule, the inner layers of the ventricular wall and the papillary muscles of the auriculo-ventricular valves are more markedly affected. Under the microscope the discolored portions show that the primitive muscular bundles are almost filled with fat droplets and granules, while the transverse and longitudinal striation has become nearly invisible.

Tab. 2. *a*



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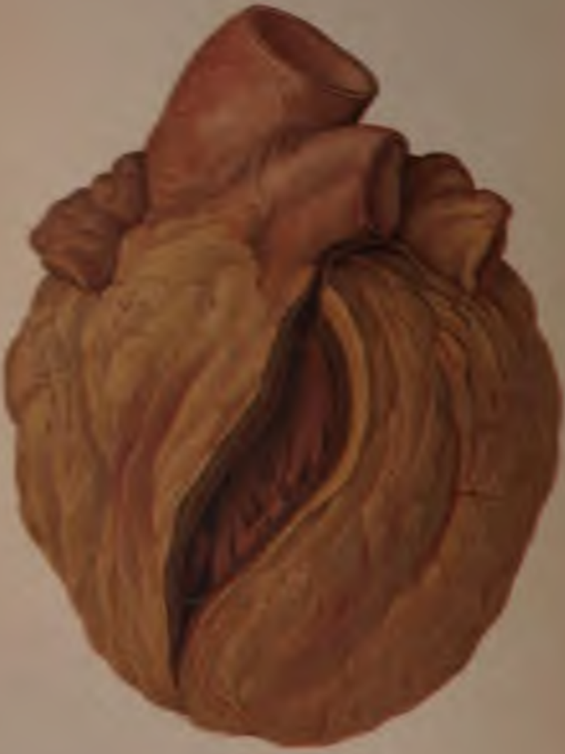


Lith.Anst.v. F. Reichhold, München

Explanation of Plate 2b.

Cor Adiposum, Adipositas Cordis.

Fatty infiltration of the subepicardial connective tissue, extending into the external layers of the heart muscle. The heart is covered with a mantle of fat through which the muscle gleams only here and there with a reddish tint. This connective-tissue proliferation containing fat is found to be most marked at the base of the heart and over the right ventricle. The limit toward the muscle is indistinct.

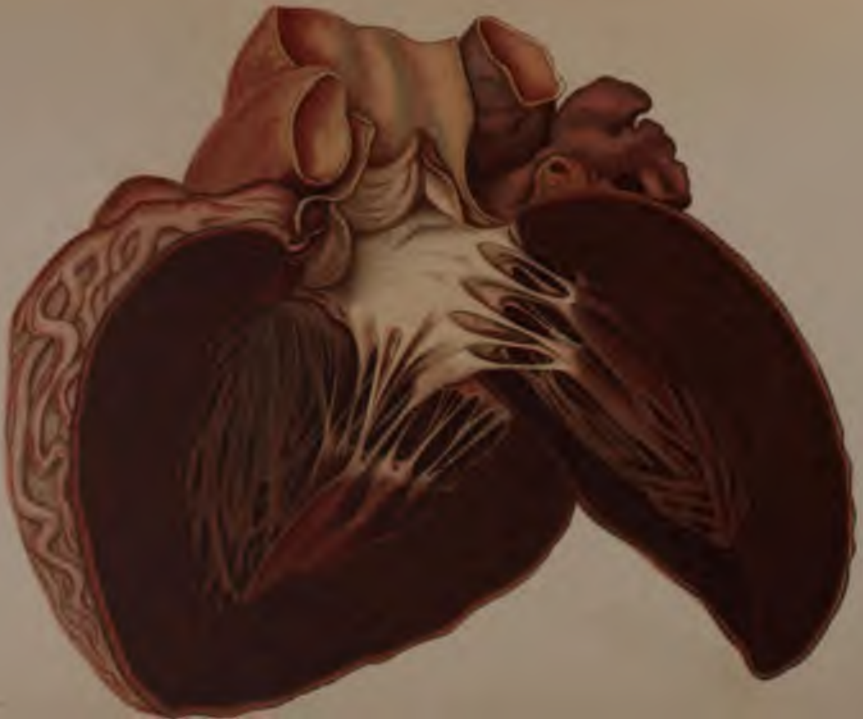
Explanation of Plate 3.

BROWN ATROPHY OF THE HEART.

The volume and weight of the heart are reduced to about two-thirds to one-half of the normal; the cavities are narrowed (concentric atrophy); the coronary arteries run a markedly tortuous course. The muscle is of a dull brownish color (almost sepia brown) and of rather firm consistence. Owing to the general anæmia, the chambers of the heart appear narrowed; but, as the muscular wall surrounds a diminished cavity, the lessened bulk is to be determined not so much by the thickness of the muscle as by the decreased weight and volume of the entire heart. In defective development of the heart (hypoplasia), which is generally associated with hypoplasia of the aorta, general anæmia, and stunted physical development, the brownish discoloration of the muscle and the characteristic tortuosity of the coronary arteries are lacking.

The heart illustrated in the plate was derived from a woman, aged seventy-two years (No. 632, 1894), who was affected with diabetes and had died of carcinoma of the pancreas with metastases in the liver, the pleura, and the left kidney. Chylous and hemorrhagic ascites was found, together with pronounced general emaciation.

Tab. 3.



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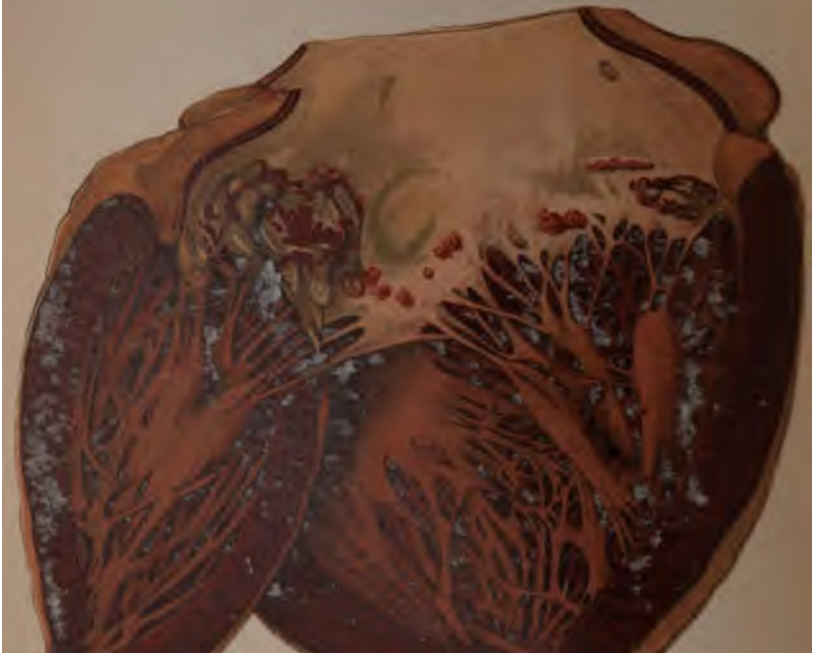


Explanation of Plate 4.

MALIGNANT MYCOTIC ENDOCARDITIS OF THE MITRAL VALVE.

On the inner surface of the median valve are found irregular, tubercular, fissured, semisoft deposits of a dull gray discolored appearance; the surface of these masses, which consist chiefly of thrombotic formations and micro-organisms, is covered with layers made up of red blood corpuscles and fibrin. The underlying tissue of the valve and the adjoining endocardium of the left auricle are in a condition of ulcerating and necrosing inflammation, and after the deposited thrombi are detached look as if gnawed into.

Corresponding to the line of closure of the mitral valve we find a larger number of reddish-gray papillary excrescences consisting mainly of young connective tissue. The valvular disease produces insufficiency and stenosis; moreover, valves thus affected very frequently give rise to emboli in the distribution of the aorta, with the formation of purulent metastases (embolic abscesses) in the brain, the spleen, the kidneys, etc. (embolic septico-pyæmia).

The heart figured in Plate 4 was derived from a puerpera, aged twenty-nine years (No. 333, 1894), dead of puerperal endometritis and septico-pyæmia. Besides the mycotic verrucose endocarditis there were embolic renal infarctions, purulent leptomeningitis, and flabby croupous pneumonia of the right lower lobe, with secondary pyofibrinous pleuritis.

Explanation of Plate 5.

RECURRENT ENDOCARDITIS OF THE AORTIC VALVES.

Chronic and Recurrent, Deforming, Fibrous and Verrucose Endocarditis of Nineteen Years' Duration.

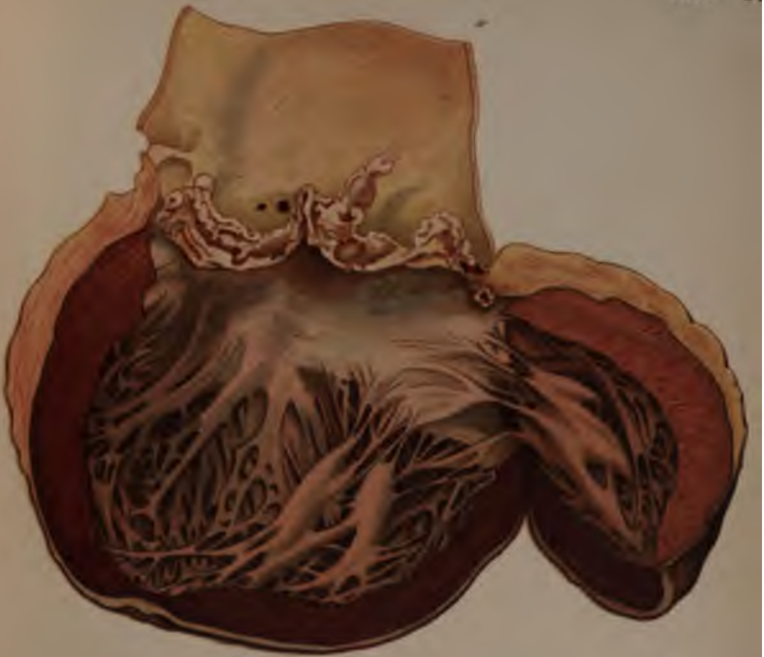
(About one-half natural size.)

FIG. *a.*—The place of the aortic valves is occupied by an irregular formation of ligamentous tissue covered partly with firm, tumid, verrucose, partly with fibrinous excrescences. The several valves have coalesced. The septum between two valves, the lateral and dorsal, has completely disappeared; upon the dorsal valve is a grayish-red, pointed, soft shred of tissue about 1 cm. in length. Owing to the truly deforming alterations, which suggest a terminated ulcerative endocarditis, the valve proves markedly insufficient and stenosed. As a secondary anomaly we find an enormous dilatation and moderate hypertrophy of the left ventricle (excentric hypertrophy), the lumen almost the size of a fist, the wall 1.2 cm. thick, pale brownish-red in color. The right ventricle is unaltered; the entire heart is evidently much widened by reason of the dilatation of the left ventricle. Associated conditions found were congested organs, no dropsy. The patient (a confectioner, aged thirty-one years, No. 384, 1894) had suffered from heart disease since his twelfth year, without demonstrable cause; articular rheumatism had never been present.

MARASMIC THROMBOSIS OF THE LEFT VENTRICLE.

FIG. *b.*—At the apex of the left ventricle is a grayish-red soft thrombus of the wall, the size of a hazelnut, with uneven surface, and whose base seems to be intimately interwoven with the trabeculæ. This terminal thrombosis of the wall was found in a patient, aged nineteen years (No. 336, 1895), dead of chronic pulmonary tuberculosis. Aside from extreme emaciation (body weight 29 kgm. = 64 lb.) there was general anæmia. No emboli had been swept from the cardiac thrombus into the arteries of the body. Otherwise, marasmic thrombi, in the form of the white and mixed varieties, are found much more often in the right ventricle and auricle than on the left side. In estimating at the post-mortem table the vital energy and activity of the heart marasmic thrombi are of special importance; in general and with reference to their pre-agonal development they are, as a rule, of less interest to the clinician. Their formation is favored, aside from weakness of the circulation, by changes in the composition of the blood, leucocytosis, and similar conditions. Their terminal occurrence, especially in numerous diseases of the heart, proves that the mechanical and physical factor of stasis, of defective circulation, plays the chief part in the formation of certain thrombi, and that lesions of the endocardium and of the intima of the vessels and alterations in the composition of the blood occupy a secondary place in such cases.

Tab. 5.



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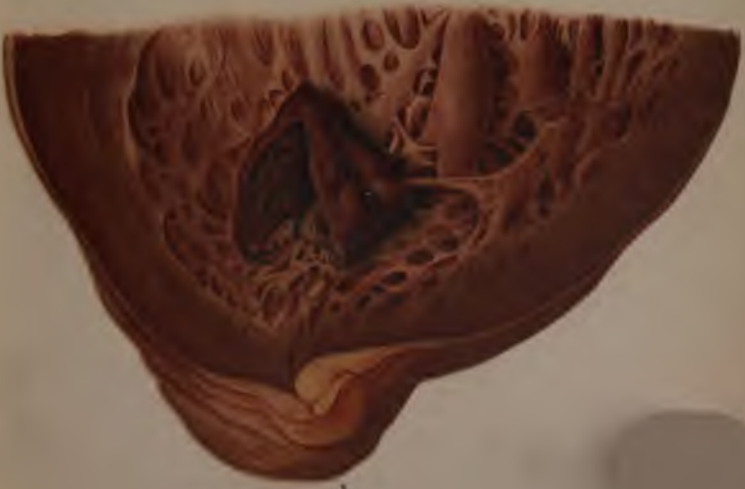

b.

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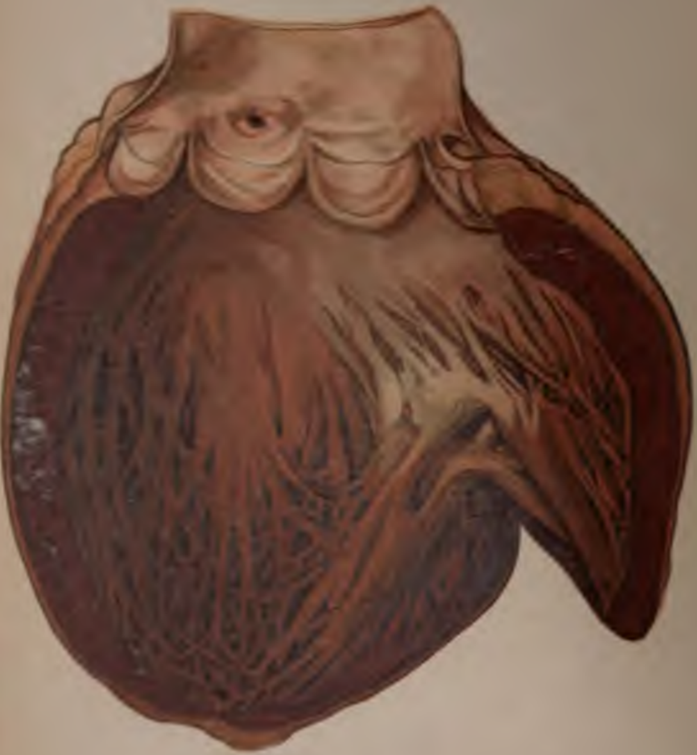

Explanation of Plate 6.

CHRONIC FIBROUS ENDOCARDITIS OF THE MITRAL VALVE, ASSOCIATED WITH CONSIDERABLE STENOSIS AND INSUFFICIENCY.

(Endocarditis Chronica Fibrosa Retrahens.)

The leaflets of the valve show a marked callous thickening; they are almost cartilaginous, excessively shortened, and form rigid, firm masses; the tendinous cords are barely recognizable. The place of the latter is taken by short cords thicker than a knitting-needle, which immediately unite the rigid valve tissue with the likewise callous indurated papillary muscles. At the circular line of attachment of the mitral valve calcareous masses are deposited. The ostium of the valve is extremely stenosed, barely admitting a thick lead pencil. Among the associated conditions were hypertrophy and dilatation of the left auricle, of the right ventricle and auricle, brown induration of the lungs (chronic engorgement), together with adhesive pericarditis and fatty infiltration of the heart.

The left ventricle was also hypertrophied, perhaps in connection with inflammatory, smooth, contracted kidneys. The patient (No. 335, 1894), aged seventy-two, from whom the specimen was obtained had suffered, moreover, during the last year of her life from a peculiar hemorrhagic diathesis which manifested itself by the fact that the least local lesion of the surface caused extensive ecchymoses.

Explanation of Plate 7.

CHRONIC FIBROUS AND CALCAREOUS ENDOCARDITIS OF THE AORTIC VALVES.

While the normal valves of the heart are delicate, translucent, and movable, the aortic valves in the present case are rigid, moderately thickened, and partly set with calcareous incrustations—the appearance of deforming, hyperplastic, and calcareous endocarditis with moderate stenosis and insufficiency of the aortic valves. The adjoining portions of the ascending aorta are similarly opacified here and there, and show points of calcification in the region of the thickened intima.

Owing to the valvular affection hypertrophy and dilatation of the entire heart developed, the left ventricle being most markedly involved (weight of the heart, 565 gm. = 20 oz., to body weight of 60 kgm. = 132 lb.). Other conditions found in the patient (No. 26, 1895), aged fifty-nine years, were congestion of the various organs, with swelling of the liver, spleen, and kidneys, slight anasarca of both feet and legs, and moderate pulmonary emphysema. Death was due to exhaustion of the heart, shown anatomically by marasmic thrombosis of the right auricle. The disease had lasted several years, during which the patient's working-power was reduced. The patient, a carpenter, had suffered much from "asthma;" in former years he had worked very hard at his trade, and had drunk brandy like other workmen, but not to excess.

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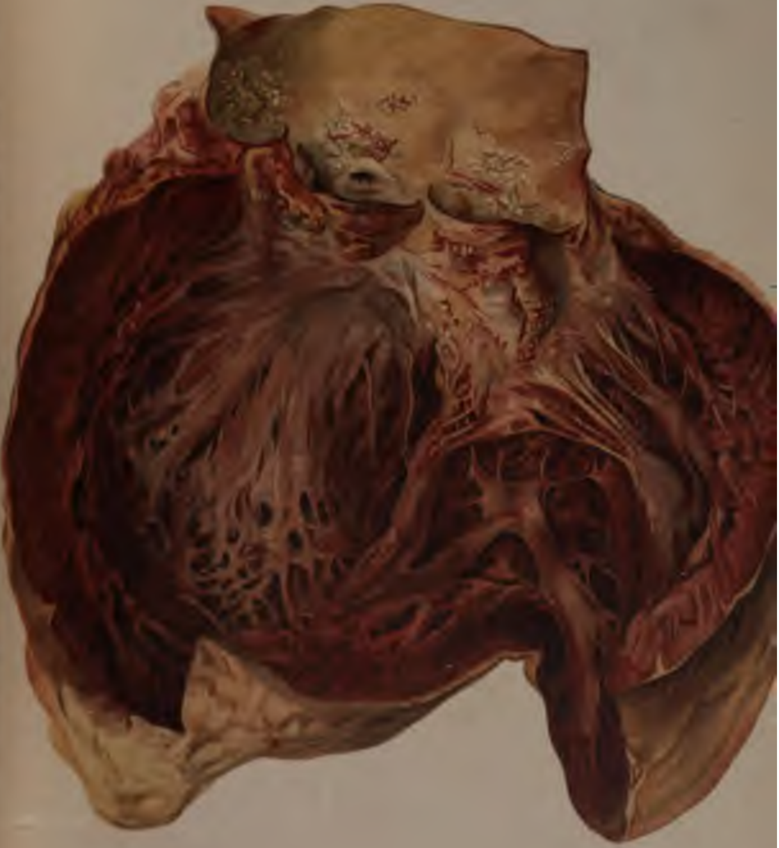

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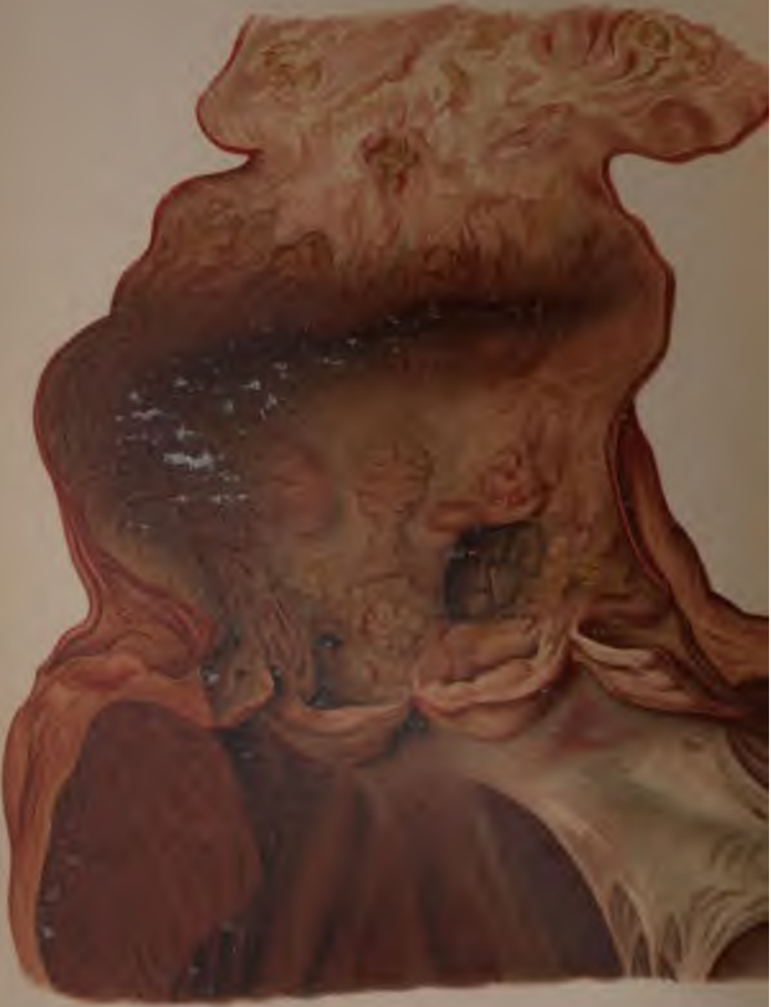

Explanation of Plate 8.

FIBROUS SCLEROSIS OF THE ASCENDING AORTA AND THE ARCH OF THE VESSEL, WITH EXTENSION TO THE AORTIC VALVES.

Insufficiency and Slight Sclerosis of the Latter.

(A small sacculated aneurism of the ascending aorta, situated immediately above the aortic valve.)

The intima of the distinctly dilated aorta is covered with irregular, flat, garden-bed-like thickenings. These fibrous elevations are variously indented on the surface and are of a grayish-white and yellowish color; besides these we may note small calcareous plates deposited, and here and there irregular jagged losses of substance with a grayish-red bottom. The deforming, chronic inflammatory process extending to the aortic valves has changed them very markedly; they show great fibrous thickening, irregular cicatricial shortening, and considerable shrinking of the valve pockets. This retraction and shortening of the valves (endocarditis fibrosa chronica retrahens), together with the dilatation of the ostium of the aorta, which is joined on one side by the dilatation of the ascending aorta and on the other side by the dilatation of the left ventricle, produced a pronounced, as it were potential insufficiency of the aortic valves (valvular and relative insufficiency). Immediately above the dorsal aortic valve is an opening the size of a dime which leads into a saccular protrusion of the wall of the aorta, the size of half a hazelnut (initial stage of a sacculated aneurism).

The patient, a hotel keeper, aged forty-two years (No. 231, 1895), had, besides the aortic disease here illustrated, marked hypertrophy and dilatation of the entire heart, especially of the left ventricle (weight, 775 gm. = 27½ oz.). Other conditions found were right hydrothorax with atelectasis by compression, congestive swelling of the liver, spleen, kidneys, and congestive catarrh of the stomach. There was no general dropsy.

Death was due to heart failure. The cause of the fatal aortic sclerosis is to be found in acquired syphilis, and perhaps in alcoholism. This presenile gummous endaortitis has a progressive malignant character and is much more frequent in men, sometimes associated with tabes. Clinically it manifests itself chiefly by aortic insufficiency, while anatomically and etiologically the aortic sclerosis represents the primary and most important disease. Syphilis and alcoholism are of special importance in the etiology.

Explanation of Plate 9.

ATHEROMA OF THE THORACIC AORTA.

(Aortitis Chronica Deformans.)

The intima of the aorta is irregularly thickened, at many points in garden-bed-like patches; it shows dull gray discolorations and yellowish spots. The flat and partly wrinkled elevations of the intima have here and there a gelatinous appearance and soft consistence. At some points the intima seems to be almost fissured and covered with irregular losses of substance, the bottom of which is formed by a dirty gray greasy mass (atheromatous fatty pulp). This intense aortic disease, which was observed in a comparatively young person (a baker aged forty-four years), is characterized chiefly by the complete absence of calcareous deposits, which are hardly ever lacking in the endaortitis of older patients, in senile and presenile arterio-sclerosis. This essentially productive and hyperplastic inflammation of the arterial wall is found mainly in men between thirty-five and fifty years of age, and is usually due to syphilis and alcoholism. It extends frequently to the aortic valves, causing insufficiency; in the present case, only the points of attachment of the aortic valves were somewhat rigid and moderately thickened.

In the case here illustrated (No. 65, 1895) we found as a co-ordinated important disease a considerable hypertrophy and dilatation of the entire heart (weight, 610 gm. = 21½ oz. to a body weight of 78 kgm. = 172 lb.), which must be considered in the main as idiopathic (alcoholic-plethoric), although the serious aortic disease acted upon the left heart in such a way as to favor the existing hypertrophy and dilatation. Other associated conditions noted at the autopsy were slight indurated moycarditis, congested organs, *i.e.*, cyanotic induration of the liver, spleen, and kidneys, congestive catarrh of the stomach, pulmonary œdema without other dropsies. The patient, who was powerfully built and well nourished, arrived moribund at the hospital; death was due to heart failure.

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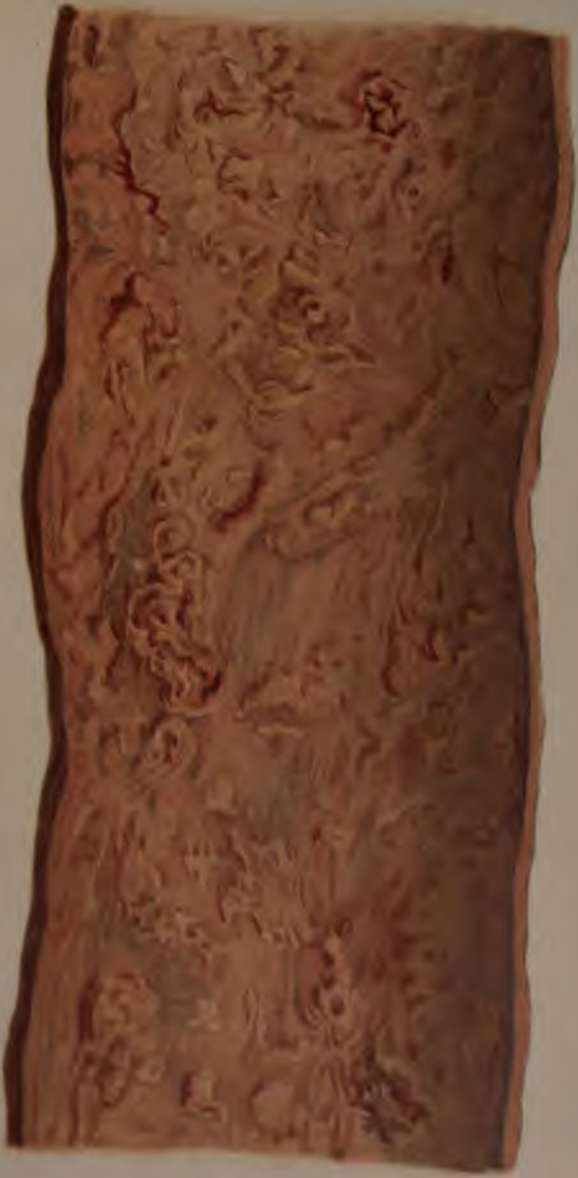
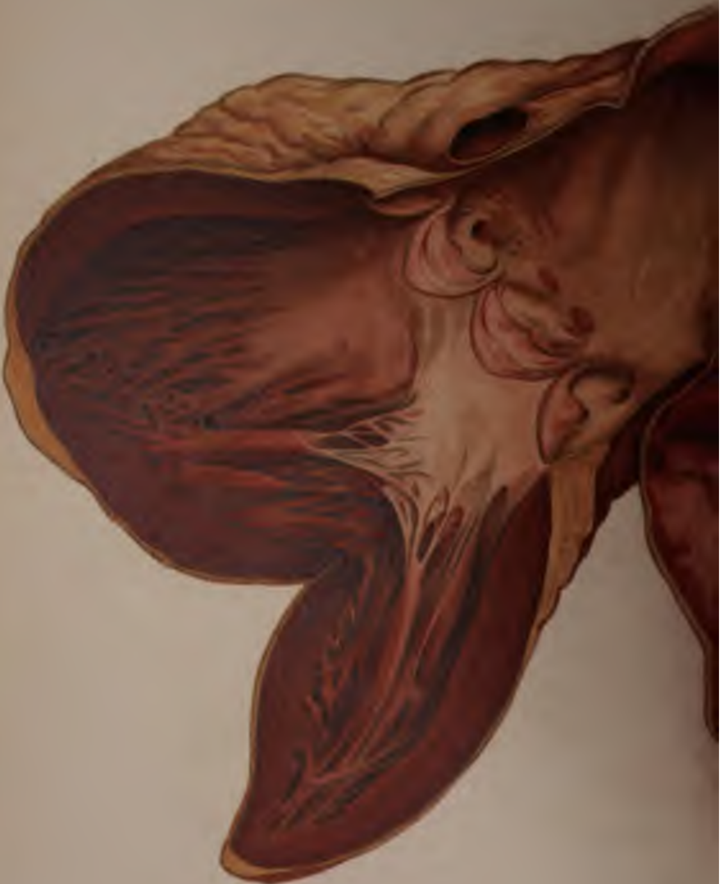
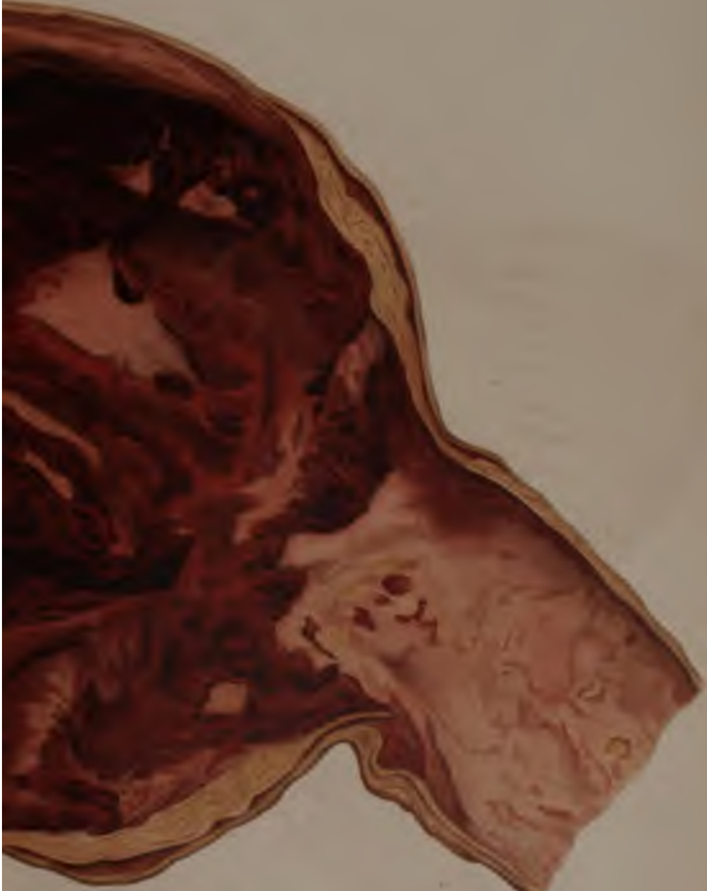






Explanation of Plates 10 and 11.

Aneurism of the Thoracic Aorta, the Size of a Fist.

(Involving the Arch of the Aorta, with the Adjoining Portions of the Ascending and Descending Aorta.)

In the anterior mediastinum, above the pericardium, was a tumid mass, having a fluctuating feel, its circumference the size of the palm of a hand. After removal of the thoracic organs the arch of the aorta with the adjoining portions of the vessel, beginning about 8 cm. = $3\frac{1}{8}$ in. above the aortic valves, was found to be considerably dilated to the size of a small child's head. The intima of the dilated portion is irregularly tubercular; some parts of the sac wall are greatly thinned, less resistant, and covered with layers of rather firm, whitish-yellow masses of fibrin. The adjoining undilated parts of the aorta show a moderate degree of sclerosis, *i.e.*, irregular ridge-like thickening, spots of opacity, and slight calcification.

The aortic aneurism has extended partly upward and to the left; the upper dorsal spinal column is somewhat eroded on the left side; the principal bronchus of the left lung is much compressed and narrowed (bronchostenosis) by the aortic dilatation.

The patient, aged fifty-three years (No. 648, 1894), was greatly emaciated; her heart was remarkably small and the thyroid gland enlarged (colloid struma). Duration of the disease about eighteen months; grave symptoms had been present for the past year. The diagnosis rested for some time between mediastinal tumor and aneurism. The etiology is quite unknown; as the patient was well-to-do (the wife of a merchant), physical overstrain and alcoholism could be absolutely excluded.

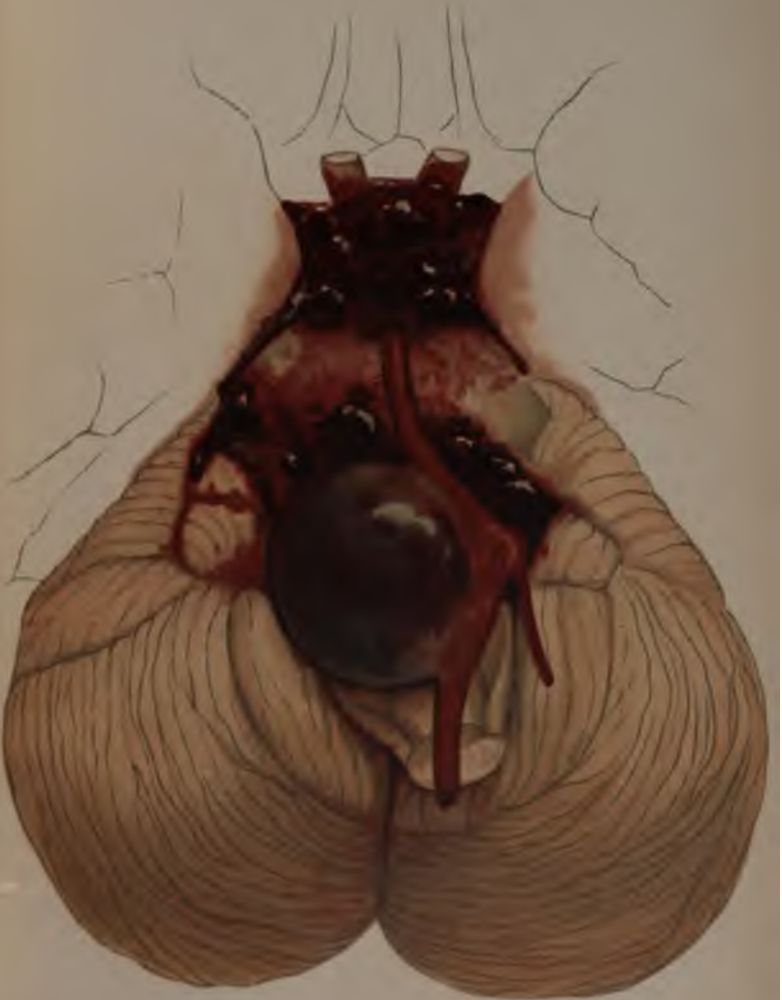
Explanation of Plate 12.

ANEURISM OF THE BASILAR ARTERY AND OF THE VERTEBRAL ARTERIES.

(Rupture, Intermeningeal Hemorrhage, Secondary Chronic Internal Hydrocephalus in a Boy Aged Seven Years.)

Over the lower part of the pons is an aneurismal dilatation, the size of a walnut, of the basilar artery. Perforation the size of a lentil, effusion of blood into the subarachnoid spaces at the base of the brain. The junction of the two vertebral arteries is involved in the aneurism. The right vertebral artery is twice the size of the left. The basilar artery springs from the anterior surface of the aneurism exactly in the median line. Nothing definite could be ascertained as to the etiology; syphilis was excluded. In the third year of life the patient had suffered an injury (a blow on the head with a hammer). Marked morbid disturbances did not appear until six days before death—Cheyne-Stokes respiration, spasm of the extremities, rise of temperature to 38 C. = 100.4° F., acceleration of the pulse, finally sopor. Among 37 cases of aneurism of the basilar artery and the vertebral arteries described in the literature, only 3 patients were between twenty and twenty-nine, one between ten and nineteen years old. (The present case is described at greater length in the paper by Willibald Oppe, entitled "Ein Fall von Aneurysma der Arteria basilaris bei einem siebenjährigen Knaben," *Münch. med. Abhandlungen*, 24 Heft, 1892.)

Tab. 12



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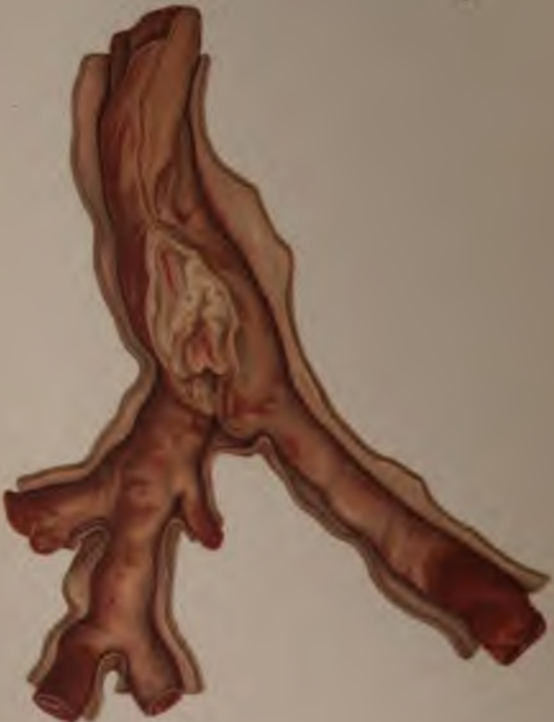

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Explanation of Plate 13.

Marasmic Thrombosis of the Uterine Veins.

The venous trunks are completely occluded by thrombi which are evidently rather recent. A considerable time after delivery, under the influence of the defective circulation, thrombi developed in the uterine veins and extended into both pampiniform plexuses, the spermatic veins, and the trunk of the inferior vena cava. In the latter the thrombus shows a light yellowish discoloration, a tough membranous consistence, and adheres firmly to the intima. In a peripheral direction both iliac and the crural veins are also thrombosed; on the left side the thrombus is undergoing puriform disintegration.

The uterus was the size of a goose's egg. The cause of death in this puerpera, aged thirty-two years (No. 708, 1895), was extreme general anæmia. There was also an incipient pleuro-pneumonia of the right inferior lobe. Death occurred five weeks after delivery.

Explanation of Plate 14.

LEUKÆMIC ENLARGEMENT OF THE SPLEEN.

Transverse section of an enormously hyperplastic spleen from a case of splenic-myelogenous and lymphatic leukæmia. The spleen (weight, 2,510 gm. = 88½ oz.) is nearly double the volume and weight of a small liver, its capsule is somewhat opacified, and it is distinctly lobulated at its sharp border. Distributed pretty uniformly throughout it we see numerous Malpighian corpuscles which are markedly swollen, mostly from the size of a hempseed to that of a pea, and are clearly differentiated from the surrounding pulp by their yellowish-white color. Among the associated conditions found in the patient, aged sixty-two years (No. 427, 1894), were lymphoid hyperplasia of the bone marrow which had a grayish-red color, and hyperplasia of the inguinal and cervical lymph glands, of the lymph follicles of the intestine, and of the mesenteric and retroperitoneal lymph glands. The latter formed a nodular bunch the size of a child's head; the individual lymph glands were swollen to the size of a hen's egg, had a semisoft medullary consistence, and a whitish-yellow color. In the liver, which was markedly enlarged, were diffuse leukæmic infiltrations of lymphoid cells. The leucocytes in the blood were greatly increased in number. Incidental findings at the autopsy were ascites, anasarca of the lower extremities, and sero-fibrinous pleuritis of the right side. Duration of the disease, whose cause is unknown, about one year.

Characteristic of leukæmia is a considerable numerical increase of the white corpuscles; in the highest grades the proportion of the red to the white blood corpuscles is as 2:3 or as 1:1, together with corresponding diminution of the red corpuscles and of the hæmoglobin contents. The white corpuscles are medullary cells (splenic-myelogenous form) or lymphocytes (splenic-lymphatic form) or both combined.

Tab. 14

Explanation of Plate 16.

LARDACEOUS SPLEEN.

(Amyloid Degeneration of the Spleen.)

FIG. *a.*—The illustration represents one-half of the organ: the spleen is somewhat enlarged, the capsule tense, and the consistence very firm; the tissue is dry, bloodless, and has a marked fatty lustre on the cut surface. The Malpighian corpuscles are barely recognizable, the trabecular framework is apparently increased.

This morbid condition was discovered in a man, aged fifty-two years (No. 88, 1895), dead of interstitial nephritis; the mucous membrane and the submucosa of the intestinal canal likewise presented considerable amyloid degeneration, the smaller arteries, capillaries, and intestinal villi being chiefly affected. Incidental findings noted at the autopsy were subacute and chronic tuberculosis of the apices of both lungs, emphysema of the lungs, moderate hypertrophy of the right ventricle of the heart, slight sclerosis of the aorta, atrophy of the liver, and general emaciation.

Besides the diffuse amyloid disease of the spleen there is a localized focal affection, the degenerative process being confined to the Malpighian corpuscles, which appear like sago grains, lardaceously gray through the parenchyma—sago spleen.

DISSEMINATED SUBACUTE TUBERCULOSIS OF THE SPLEEN.

FIG. *b.*—Scattered through the organ, which is markedly enlarged, is a considerable number of yellowish nodules of an irregular form, ranging in size from a pin's head to almost that of a pea. The parenchyma of the hyperplastically enlarged spleen is of a dark brownish-red and bluish color, moderately firm, and rich in blood. This form of splenic tuberculosis is found most frequently in children and young persons as a partial phenomenon of a general tuberculosis, usually starting from the lungs or from cheesy lymph glands. Acute miliary tuberculosis is more frequently discovered in the spleen; in that case the organ is several times the normal size, and scattered through it are numerous barely visible submiliary and miliary nodules of a gray appearance through the parenchyma; in the centre of each may be recognized a punctiform whitish or yellowish opacity, and in their initial stage they may be mistaken for the normal Malpighian corpuscles. As a rule the spleen, similar to the lymphatic glands, is very liable to metastatic tuberculosis, while primary tuberculosis is hardly ever observed.

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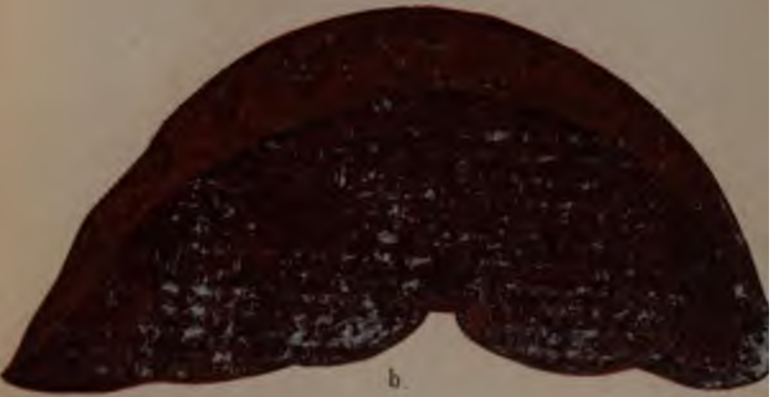

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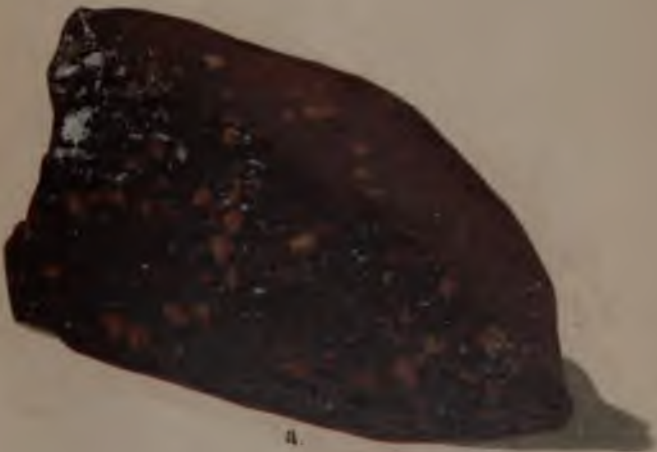

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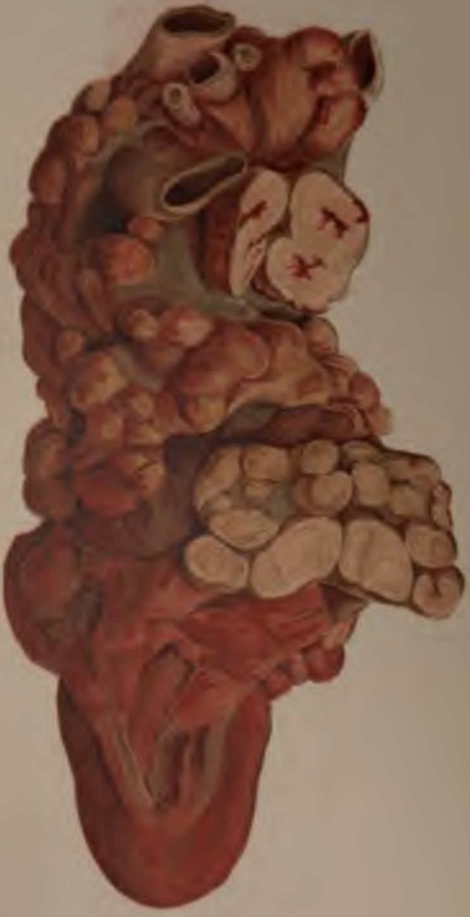

Explanation of Plate 17.

CHEESY TUBERCULOSIS OF THE CERVICAL AND INTRA-THORACIC LYMPHATIC GLANDS.

(Tuberculous Scrofulosis of the Lymphatic Glands.)

The submaxillary and cervical lymphatic glands as well as those of the mediastinum and the root of the lung are enlarged throughout, usually from the size of a pea to that of a cherry, and united into bunches. The swollen glands are firm to the touch, the tissue of many gleaming yellowish through the tense capsule. On the cut surface the gland tissue appears changed into a friable, crumbly, cheesy mass of a light yellowish and whitish color; here and there the cut surface resembles a raw potato. The mesenteric glands have undergone a similar change.

The specimen was obtained from a child, aged four months (No. 379, 1895), whose mother had succumbed to a pulmonary tuberculosis of extremely rapid course ("hasty consumption"). Aside from the glandular tuberculosis the cause of death and chief morbid changes found were acute general miliary tuberculosis of the lungs, liver, and spleen, which had obviously resulted from the primary cheesy tuberculosis of the lymph glands by way of hæmatogenous auto-infection.

Among the incidental conditions found in the child, which was extremely emaciated and very poorly developed (body weight barely 2.25 kgm. = 4⅞ lb.), were caries of the right petrous bone, general furunculosis, and catarrhal enteritis.

This was a characteristic case of primary glandular tuberculosis (tuberculous scrofulosis), in which the poison had penetrated with the air or with the food or with both from the mucous membranes (of the head, the neck, and the lungs) into the lymph channels and lymph glands. An intra-uterine, hereditary infection is possible but not probable, since the entire morbid picture may be ascribed without difficulty and more simply to infection after birth.

Explanation of Plate 18.

ADENOMA OF THE THYROID GLAND. COLLOID STRUMA

Transverse section through the thyroid gland, which i
enormously enlarged to the size of nearly two fists. Th
gland completely covers the anterior and lateral cervic
region, begins on both sides in the neighborhood of th
mastoid process, and reaches from the chin to the man
brium sterni. The gland is rather firm in consistenc
and distinct lobulation can be recognized laterally. A
ter removal of the cervical organs it is found that th
tumor surrounds anteriorly and laterally the larynx an
trachea, leaving posteriorly only a narrow groove i
which the œsophagus is embedded. From the thyroi
cartilage downward to within a few centimetres abov
the bifurcation the larynx and trachea appear flatten
like a sword sheath. The enlarged thyroid gland is har
to cut, the divided surface is of a grayish-red and gray
ish-yellow color; all over it may be seen embedded sag
like, yellowish, translucent colloid masses.

Other conditions found in the patient, aged sixt
years (No. 99, 1895), who was moribund when receiv
at the hospital, were a considerable hypertrophy an
dilatation of the right ventricle (weight of the heart, 4
gm. = 15 oz.), ascites, anasarca of the lower extremiti
and of the subcutaneous tissue of the lateral and posteri
portions of the trunk. Incidental findings were obsole
tuberculosis of both lungs, atrophy of the liver an
spleen, and general emaciation. The immediate caus
of death was not the marked stenosis of the trachea bu
the disease of the heart. The combination of goitr
cardiac hypertrophy, and exophthalmus produce durin
life a special morbid entity known as Basedow's
Graves' disease.

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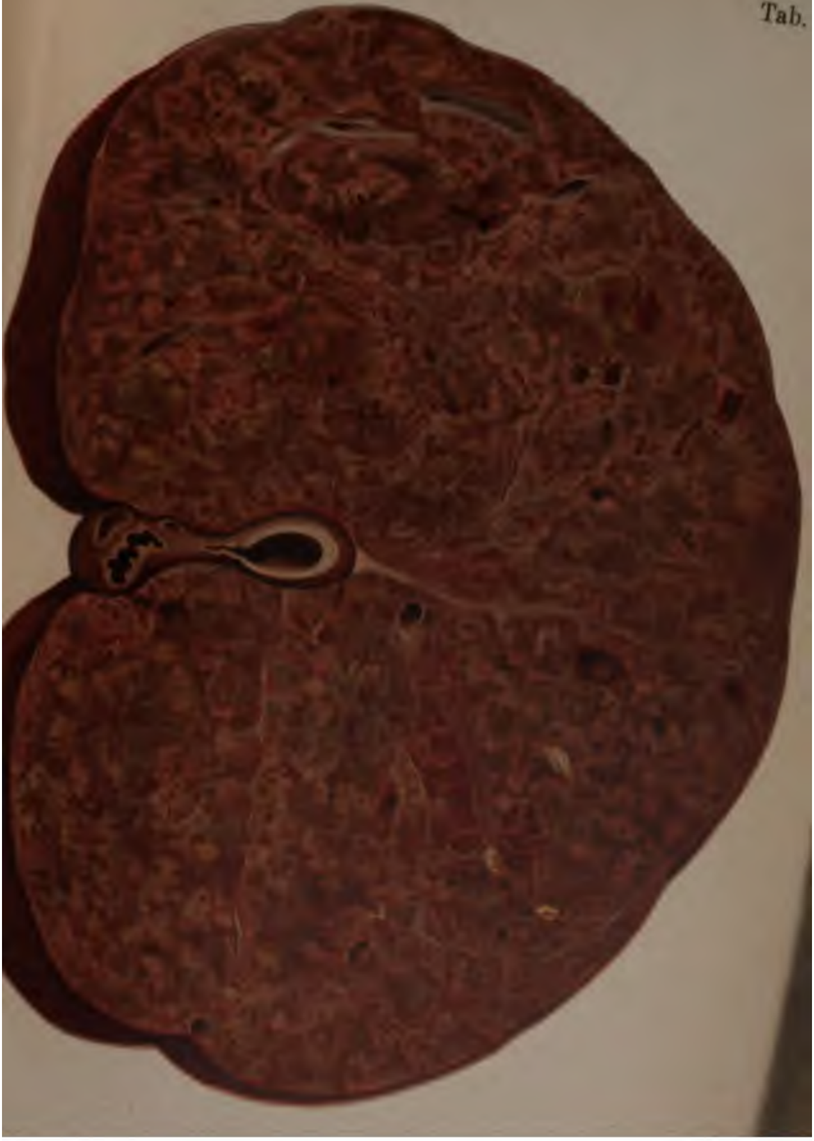

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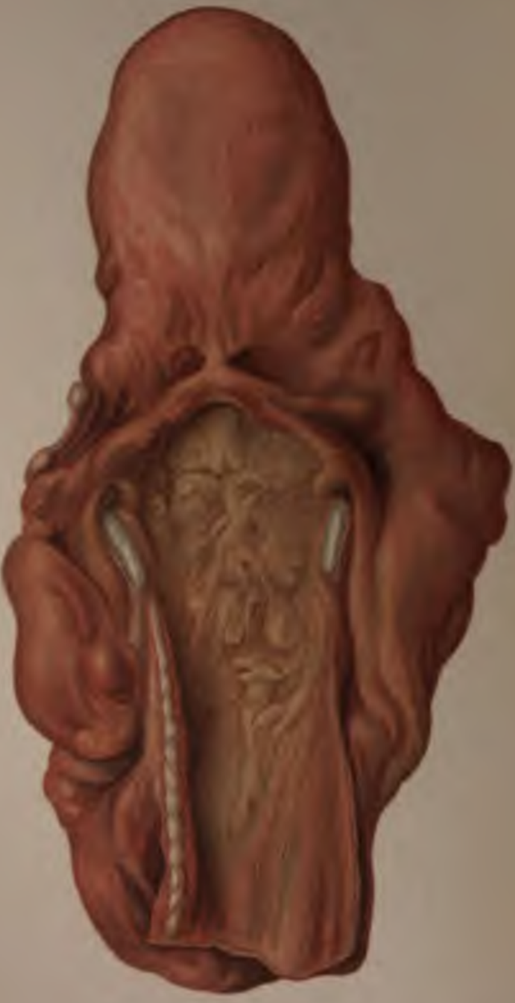

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Explanation of Plate 19.

DIPHTHERIA AND CROUP OF THE LARYNX AND TRACHEA IN A CHILD.

The mucous membrane of the larynx and trachea is covered with a yellowish-gray pseudo-membrane; the latter appears somewhat folded and looks as if it could be easily detached. The mucosa itself is reddened and swollen as is that of the pharynx, which is in a state of acute inflammation but is free from diphtheritic deposit.

The fatal issue was due to bilateral purulent bronchopneumonia.

Explanation of Plate 20.

Ulcerative Tuberculosis of the Larynx wit Necrosis of the Cricoid Cartilage.

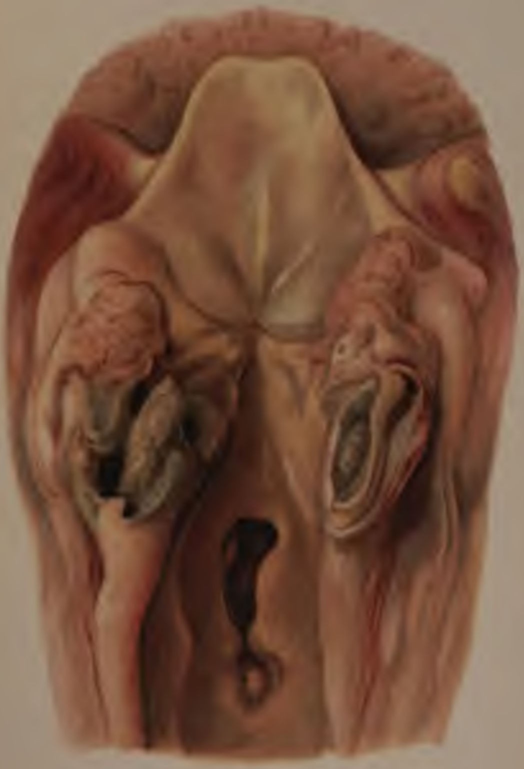
In the left posterior wall of the larynx is a la
ulcerated defect; in the depth of the excavated openi
which is almost as large as a cherry, we observe at
left side a detached cheesy and calcareous sequestru
the interior wall of the cavity has a corroded aspect a
is covered with a purulent discolored secretion.

The mucosa at the entrance of the larynx, especia
about the aryepiglottic folds, is greatly swollen and
lumen of the aditus is considerably narrowed in con
quence. The rest of the mucous membrane of the lary
is moderately thickened, smooth, and pale in color.

During life the symptom complex was so peculiar t
cancer of the larynx was suspected for some time.

Directly beneath the larynx in the first part of
trachea is a gaping, slit-like loss of substance caused
tracheotomy. Besides the serious ulcerative and necr
affection of the larynx there were found in the patie
who was forty-seven years old, chronic pulmon
tuberculosis and ulcerating tuberculosis of the colon.

Tab. 20.



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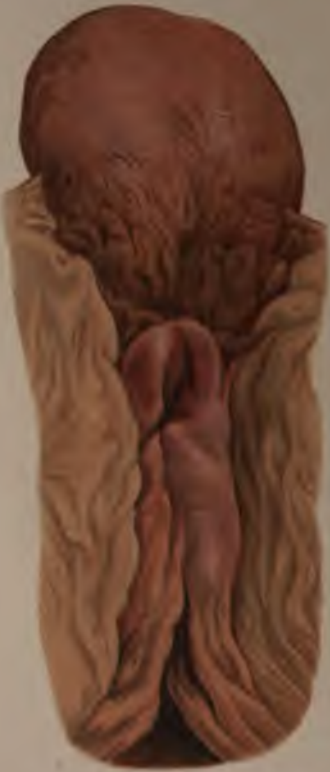

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Explanation of Plate 21.

ACUTE ŒDEMA OF THE GLOTTIS.

(Pharyngo-laryngitis Erysipelatosa Acutissima.)

The mucous membrane at the aditus laryngis, especially that of the epiglottis and the aryepiglottic folds, is markedly swollen and reddened, and in consequence thereof the entrance of the larynx is almost occluded. The base of the tongue and the tonsils show a similar condition of the mucous membrane: a bluish-red discoloration and considerable swelling.

Death was due to asphyxia after a brief indisposition, and occurred on the way to the hospital; the patient having exhibited himself the evening before as a "fat man."

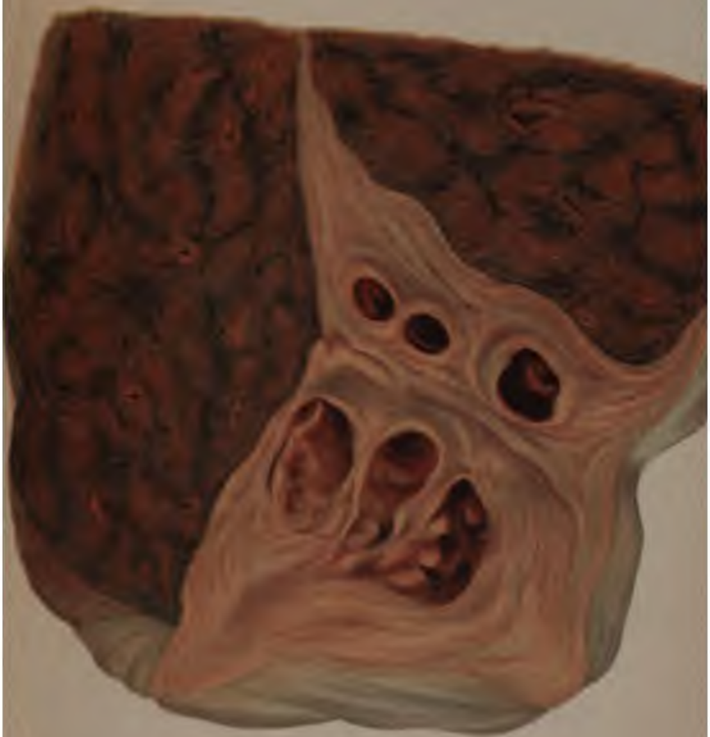
The subject was extremely obese by reason of an inherited tendency; the body weight in the fourteenth year was 90 kgm. = 198½ lb.; at the time of death, 201 kgm. = 443 lb. The acute fatal inflammatory œdema was due to erysipelatous infection (No. 92, 1894).

Weight of the heart, 1,090 gm. = 38½ oz. or 1:184; liver, 3.5 kgm. = 7 lb. 11½ oz.; spleen, ½ kgm. = 17½ oz.

Explanation of Plate 22.

BRONCHIECTASIS AND CIRRHOSIS OF THE LUNG.

The tissue at the apex of the lung over an area size of half a fist is sclerosed and crossed by irregul and markedly dilated bronchioles. The walls of latter are thickened and pass imperceptibly into the rounding cirrhotic tissue, which exceptionally does present the usual slaty discoloration. The conden portion is sharply demarcated from the surround healthy lung tissue, only a gradually diminishing c nective-tissue cord is seen extending toward the roo the lung.



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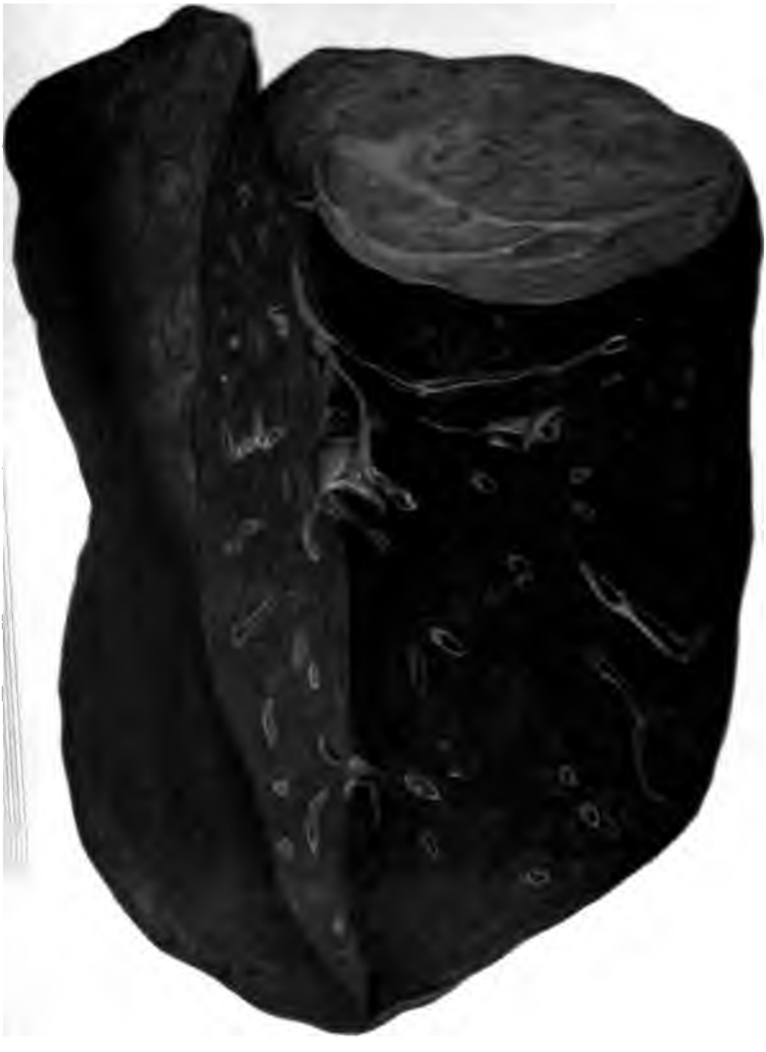

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Explanation of Plate 23.

CHRONIC PULMONARY ENGORGEMENT.

(Brown Induration.)

The lung has a dark brownish-red color, is condensed, and of a firm, tough, almost fleshy consistence. The contained air is diminished; some rusty brown fluid escapes from the cut surface.

The cause of the engorgement was at the autopsy found to be a recurrent verrucose and fibrous endocarditis of the mitral and aortic valves associated with stenosis and insufficiency; the tricuspid valve was similarly affected. The valvular disease was caused by articular rheumatism (No. 697, 1895).

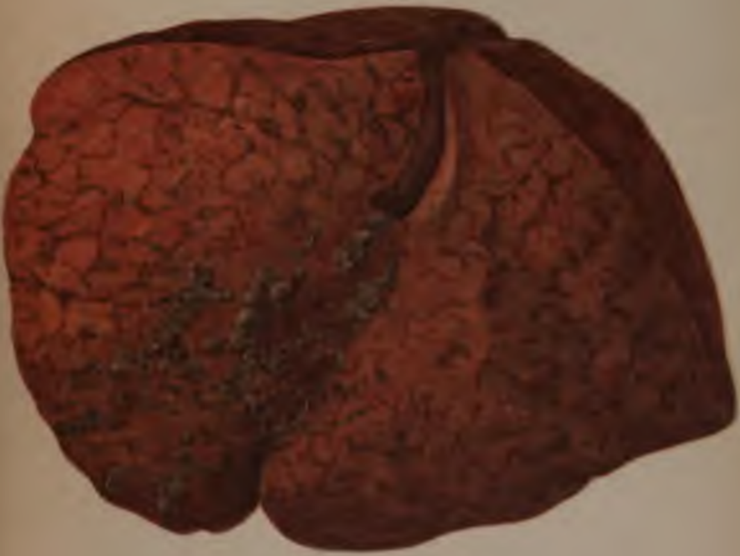
Secondary conditions found were hypertrophy and dilatation of the right and left heart and congested organs: engorgement of the spleen, nutmeg liver, cyanotic induration of the kidneys, congestive catarrh of the stomach, and general dropsy. The patient was a woman aged twenty-three years.

Explanation of Plate 24.

INTERSTITIAL PULMONARY EMPHYSEMA IN A CHI

At the surface of the lung, in the subpleural con
the tissue, we find crowded together in one place
[illegible] bubbles which are clearly demarcated f
the surrounding normal lung tissue. The air bubbles
[illegible] arranged in rows, here and there like a ros
In the parenchyma of the lung (no visible in the pl
similar strings of air bubbles are found deposited, mo
in the interlobular connective tissue.

Tab. 24.



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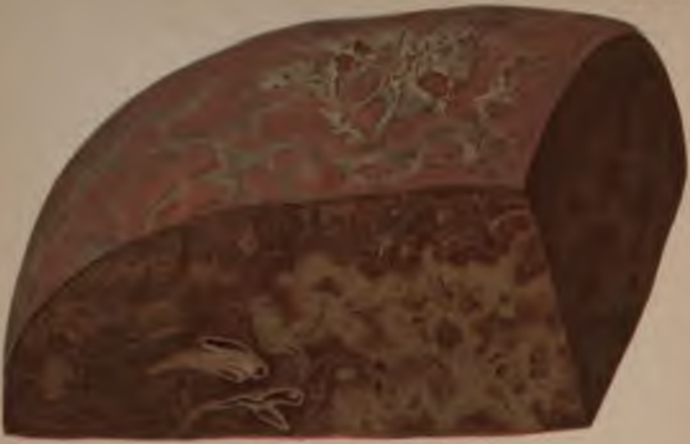

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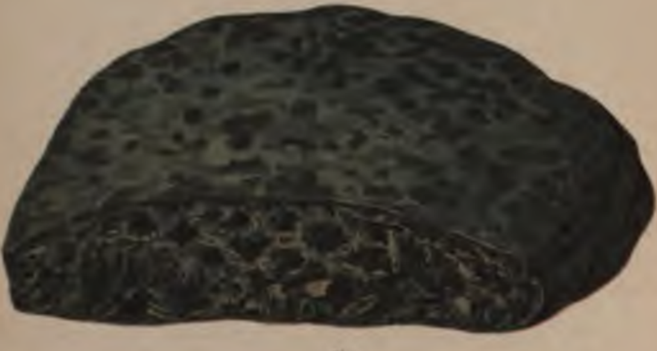

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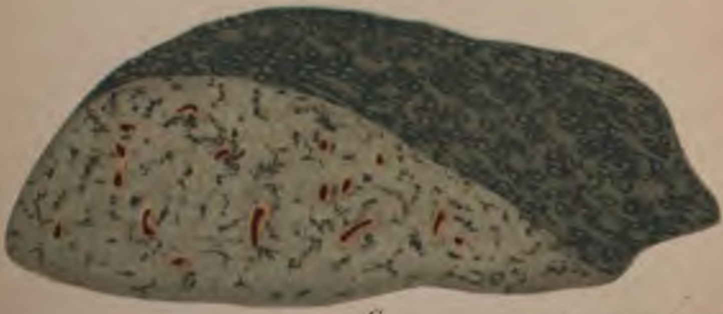

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Explanation of Plate 25.

DISEASES OF THE LUNG CAUSED BY INHALED DUST.

(Pneumonoconiosis.)

FIG. *a.*—Red Iron Lung, Siderosis.—The lung tissue is deficient in air, of a very firm, board-like consistence, and of a reddish-brown color. Through the transparent pleura and in the parenchyma we see irregular, pale gray, fibrous condensed portions which are almost free from pigment. The lung was derived from a working-woman who for several years was employed in a Nuremberg factory in coloring blotting-paper by dry-rubbing it with colcothar (ferric oxide). (The specimen was presented to the Pathological Institute at Munich by Dr. Merkel, of Nuremberg.)

FIG. *b.*—Coal-Miner's Lung, Anthracosis.—A portion of a lung which is colored throughout pretty uniformly a slaty black. On the pleural side and on the cut surface we observe the interlobular connective tissue to be somewhat lighter colored, a slaty gray. The pulmonary parenchyma is condensed. The specimen was derived from a coal-miner who had died in his fiftieth year of a compound fracture of the thigh and had worked in a mine for about thirty-five years.

FIG. *c.*—Stone Lung, Chalicosis.—The surface of the lung is of a steel-blue color, with distinct spots, and imparts to the hand almost the feel of a grater. Beneath the pleura and in the parenchyma are numerous scattered firm nodules, ranging in size from a pin's head to a hempseed, many of which have a faint slaty gray areola. Associated with the multiple nodules is a certain degree of anthracosis (chalicosis anthracotica). The pale color of the tissue, which is particularly marked on the cut surface, has been artificially produced by preservation of the specimen in alcohol. The patient was a stone-cutter.

Explanation of Plate 26.

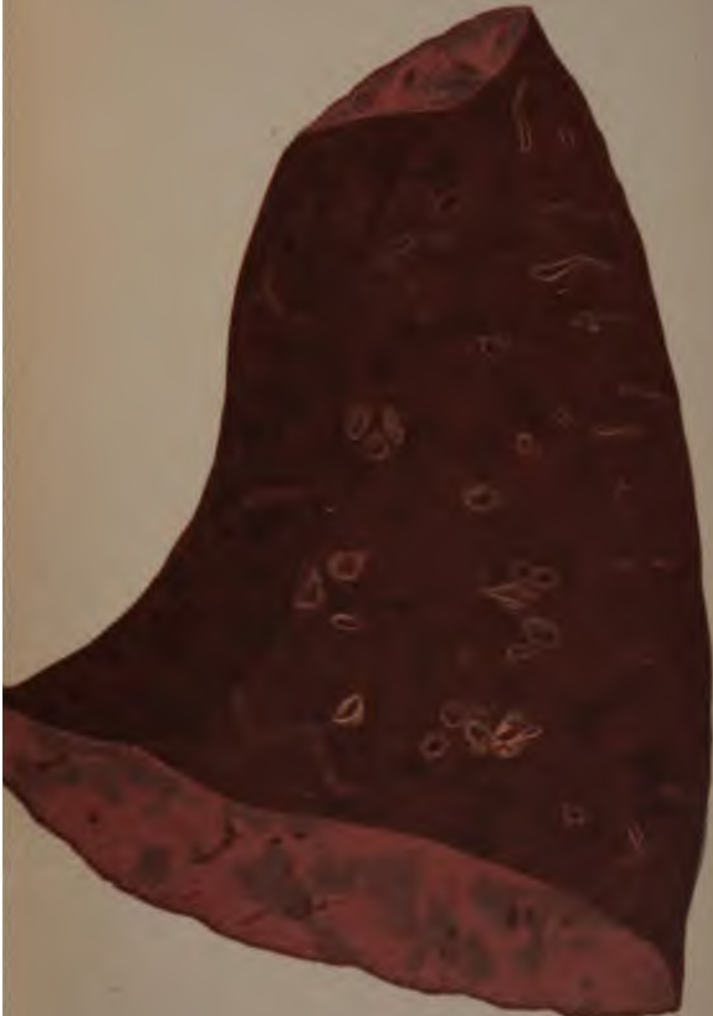
CROUPOUS PNEUMONIA.

Stage of red hepatization.

The lower lobe of the right lung is of a uniform most liver-like consistence and brownish-red color; cut surface is finely granular, and the air is expe On pressure a considerable amount of a gray p matter escapes from the large and medium-sized bronc

The patient (No. 677, 1895) was of delicate build badly nourished, weight 42.5 kgm. = 94 lb., and cumbed about the sixth day of the disease.

Tab. 26



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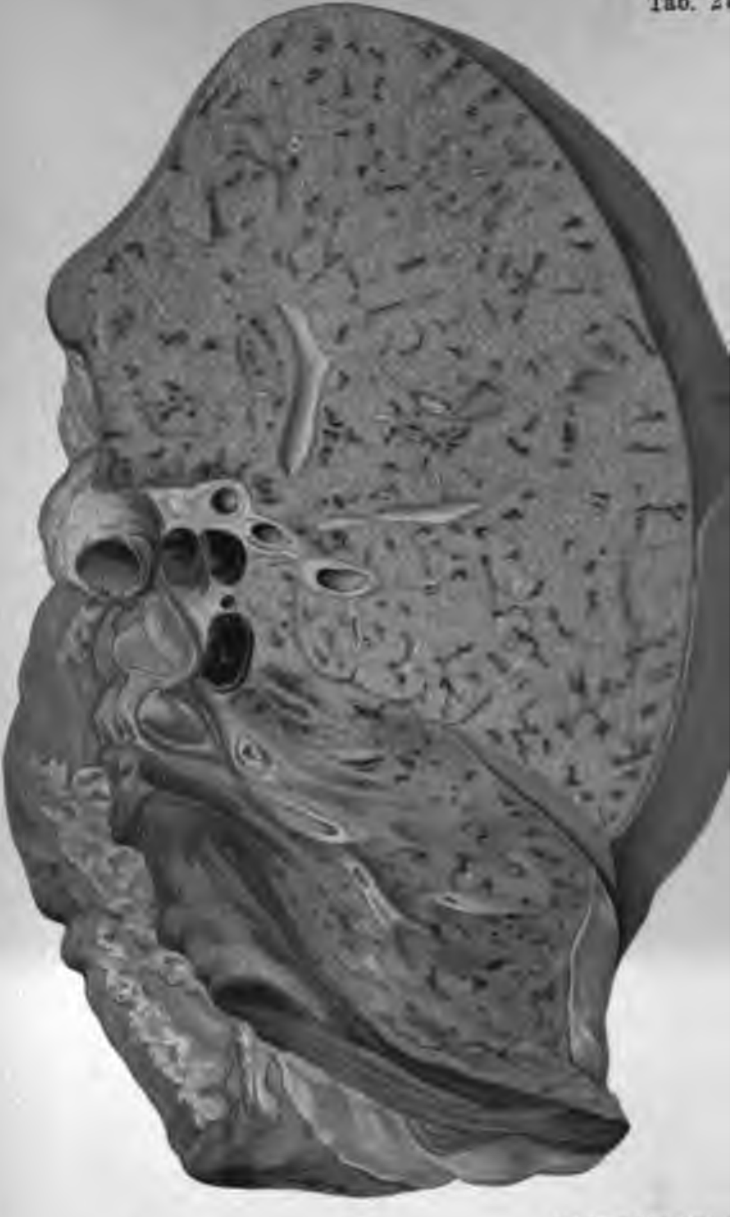

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Explanation of Plate 27.

CROUPOUS PNEUMONIA.

(Stage of gray hepatization [seventh day].)

Lobar croupous inflammation of the right superior **lobe** and of the adjoining portions of the median and inferior lobes, in the stage of gray hepatization. Death **on the** seventh day after the occurrence of the initial **rigor.** The infiltrated portions of the lung are of a firm, **liver-like** consistence, completely devoid of air, weight **1,070 gm.** = 3¾ lb.; the cut surface is of a dull **reddish-gray** color, finely granulated, with embedded slaty **blackish** oblong spots (anthracosis of moderate degree). **To the left** below and to the right externally we observe **upon the** pleura a yellowish-white and in part reddish, **loose pseudo-membrane** composed of leucocytes and fibrin. **The pleural** sac contains ¼ litre (½ pint) of a sero-purulent exudate. At the root of the lung several blackish **peribronchial** lymph glands are embedded. Aside from **the fatal** pneumonia and pyofibrinous pleurisy there **were found** incipient pericarditis (reddening and velvety **opacity** of the epicardium over the auricles), together **with** verrucose chronic endocarditis of the mitral and **aortic** valves. The patient, aged twenty-three years (No. 647, 1894), a waitress, had from her tenth year on **suffered repeatedly** from articular rheumatism.

Explanation of Plate 28.

TUBERCULOSIS OF THE APEX OF THE LUNG.

FIG. *a*.—In the apex of the lung is a cherry-s yellowish focus broken down in the centre; a hemps sized loss of substance in the centre communicates a bronchus (initial stage of the formation of a cavi Some distance farther downward is a similar yello cheesy focus of a circular form. In the surroun aërated tissue, particularly to the left, are several s gray firm nodules, some of them in partial commen caseation.

ACUTE MILIARY TUBERCULOSIS OF THE LUNG.

FIG. *b*.—Scattered through the lung tissue, along several larger, irregularly shaped cheesy foci, is a siderable number of miliary tubercles; toward the and above similar nodules are visible through the ple

The patient was a girl, aged four months (No. 1895), whose mother had died of hasty consumpt Besides the acute and subacute pulmonary tubercul there were found miliary tuberculosis of the liver spleen, which had started from a more advanced ch tuberculosis of the cervical, intrathoracic, and me teric lymph glands. Incidental conditions found at autopsy were caries of one petrous bone and furuncul of the skin.

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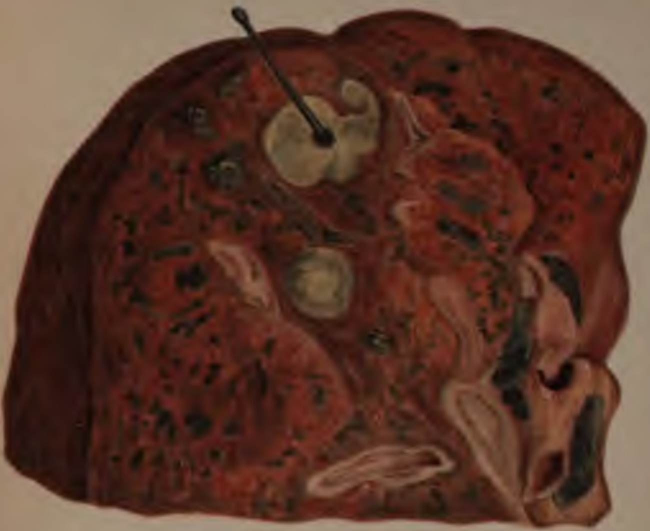

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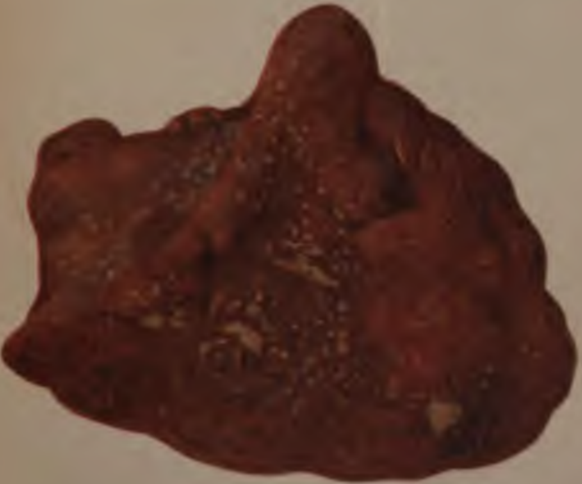

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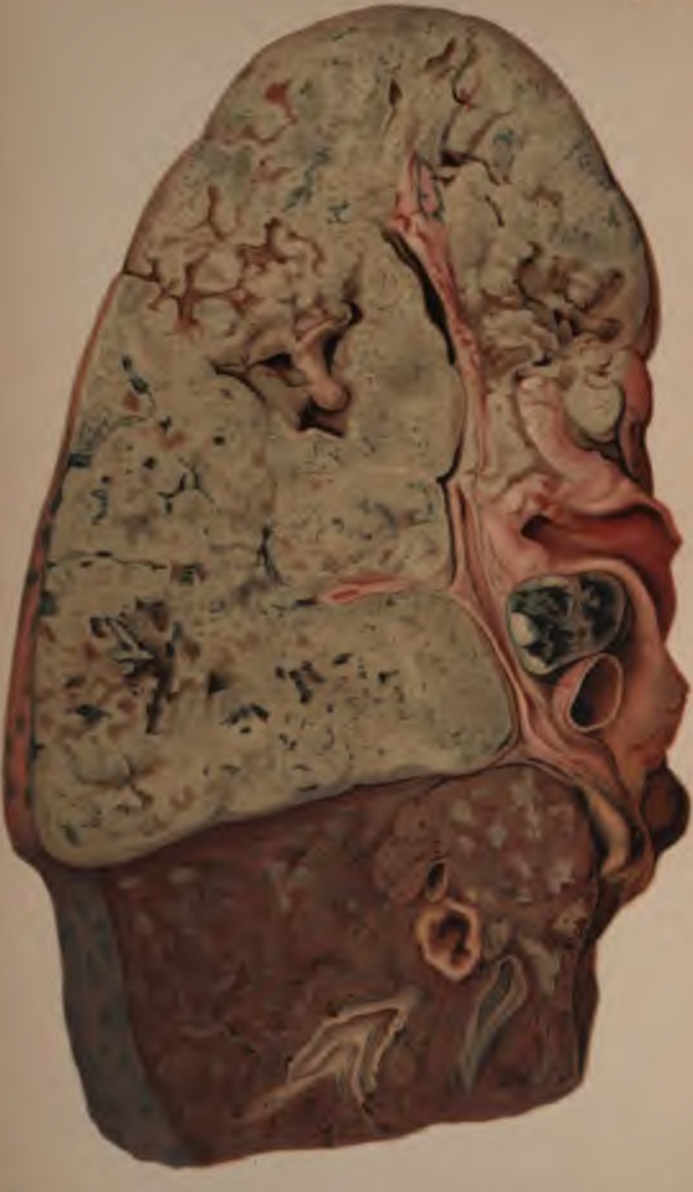
Tab. 29.



Lith.Anst.v. F. Reichhold, München.

Explanation of Plate 29.

SUBACUTE CHEESY TUBERCULOSIS OF THE LUNG.

Nearly the whole upper lobe is of a firm, liver-like consistence; the tissue is devoid of air and hard to cut; the surface of the section has a pretty uniform yellowish-white color and is friable and crumbly. Near the top, between the coalescing cheesy infiltrated lobules, we still observe some reddish colored traces of aërated tissue; at other points there is besides a commencing ulcerating and cavernous disintegration.

In the lower lobe there are only some isolated, irregular cheesy foci, ranging in size from a hempseed to half a bean. At the root of the lung is a slate-colored peribronchial lymph gland in the parenchyma of which are embedded some cheesy tuberculous foci.

Explanation of Plate 30.

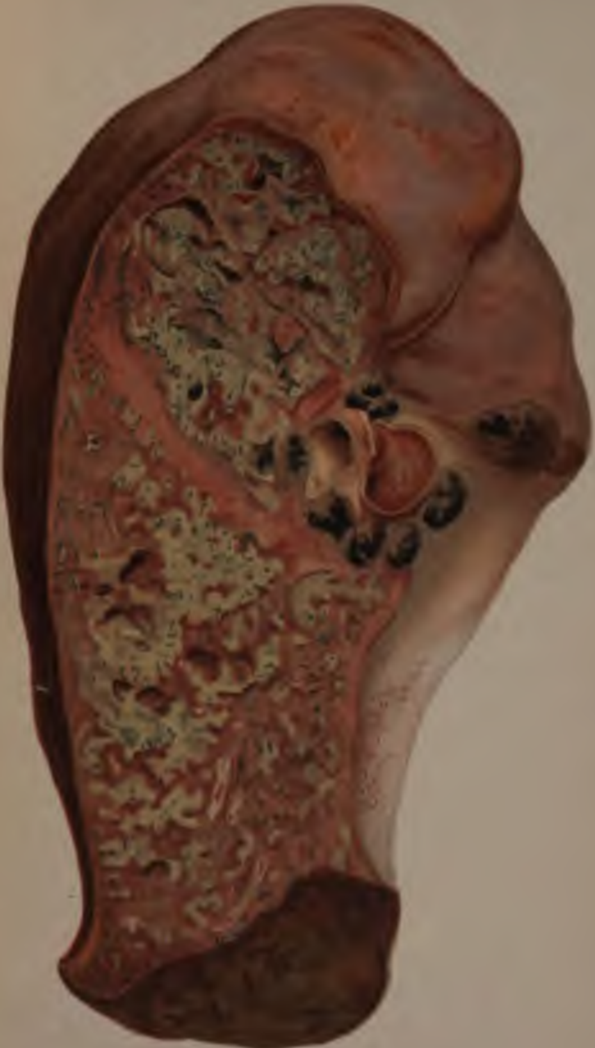
SUBACUTE AND CHRONIC TUBERCULOSIS OF THE L

Scattered in the reddish-colored, rather anæmic aërated lung tissue we find a considerable numbe mostly irregular, grayish-white foci, here and sprinkled with slaty specks; these are of a fr cheesy consistence and sharply demarcated from surrounding substance; the smallest of them, ra in size from a pin's head to a hempseed, permit o recognition in their centre of the lumen of a bronc (tuberculous peribronchitis and bronchopneumo The larger foci, many of them due to the confluen smaller patches, exhibit, especially in the upper por of the lung, occasional central disintegration, comn ing cavity formation; the smaller, irregular cav filled with cheesy pus and detritus, communicat many points with the bronchia.

At the root of the lung we observe slaty discol sooty peribronchial lymphatic glands in which ma recognized here and there beginning caseation.

Other conditions found in the patient, aged tw seven years (No. 628, 1894), were tuberculous adh pleurisy, secondary ulcerative intestinal tubercu and pronounced general emaciation.

Tab. 30.



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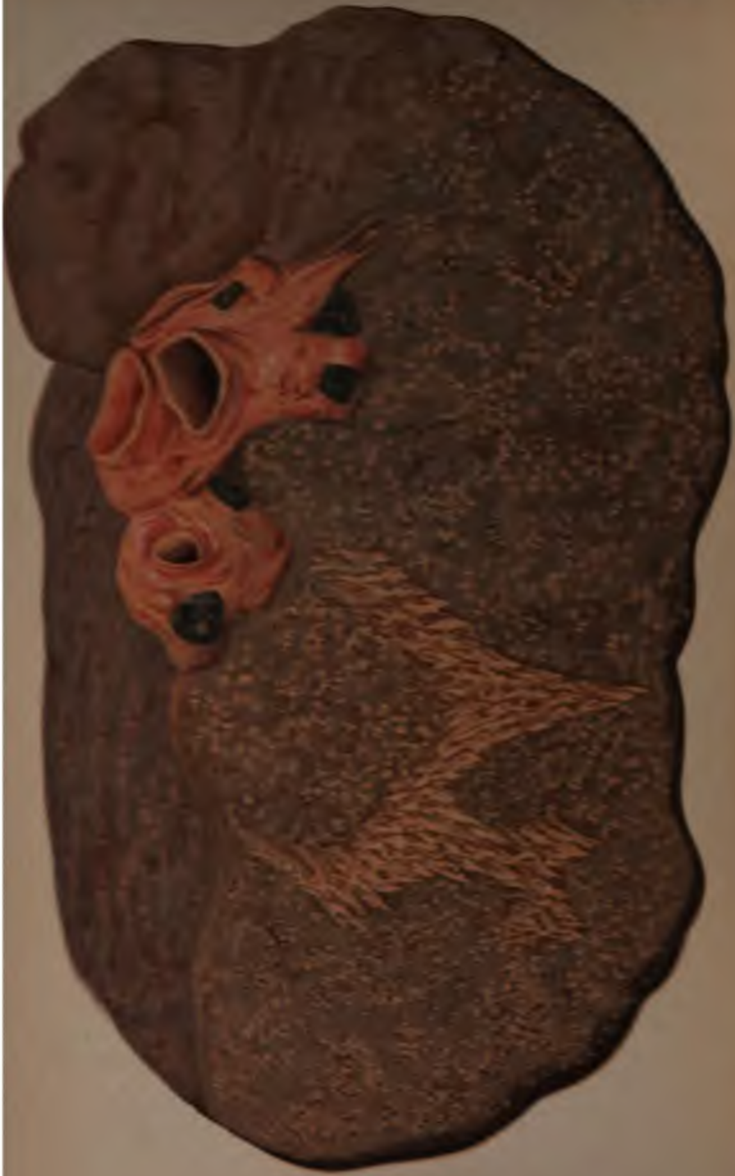


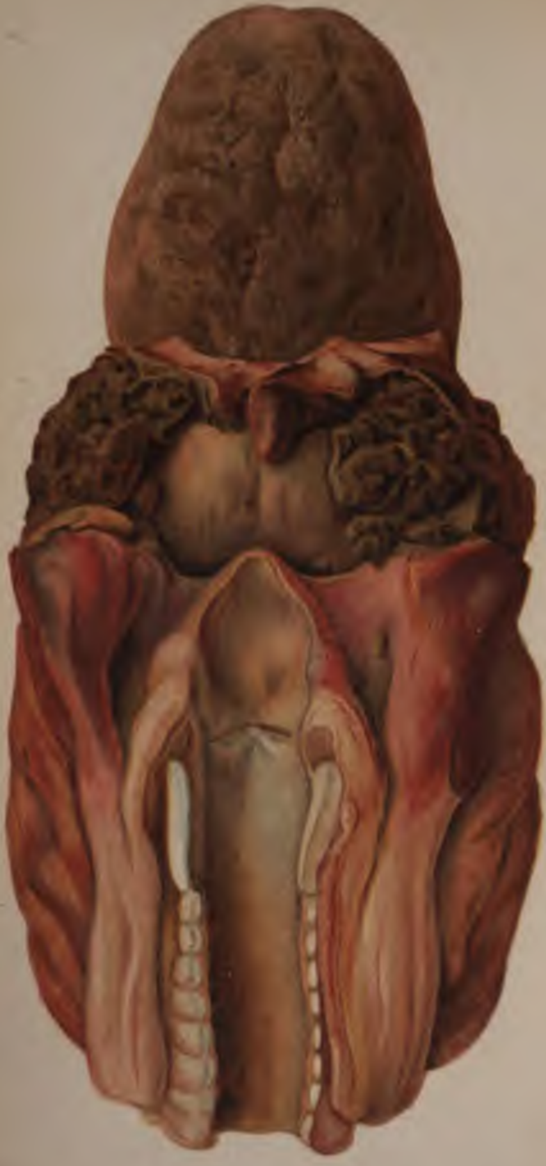
Lith. Inst. v. F. Reichhold, Mün

Explanation of Plate 31.

ACUTE TUBERCULOUS SERO-FIBRINOUS AND HEMORRHAGIC PLEURISY.

The left pleural sac contains about three litres (quarts) of a sero-sanguinolent fluid. The lung is greatly diminished in size. The pleura is covered with delicate, reddish discolored, fibrinous deposits. After their removal we observe numerous miliary gray nodules (miliary tubercles) on the pleura. At the apex of the lung—probably the starting-point of the tuberculous pleurisy—is a cheesy tuberculous focus which is evidently older. In the parenchyma of the lung (visible through the pleura to the left posteriorly) miliary tubercles are also present in moderate quantities. Other lesions found in the patient, aged fifty-nine years (No. 7, 1895), were chronic fibrous endocarditis of the aortic valves, cirrhosis of the liver, induration of the kidneys and spleen, with considerable swelling of the latter.

Tab. 33.



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Explanation of Plate 33.

GANGRENOUS DIPHTHERIA OF THE TONSILS.

The tonsils on both sides are enlarged to the size of a hazelnut; their surface is irregularly fissured, their odor is offensive, and their color a dirty brownish-yellow. On section the parenchyma of the tonsils is found in condition of gangrenous necrosis. The surrounding mucosa is deep red and swollen, the uvula in particular is greatly thickened and has a juicy lustre. The surface of the tongue has an opaque brown color. The larynx is intact.

Incidental lesions: multiple ecchymoses of the gastric and intestinal mucosa and of the external skin; lobular pneumonia. The patient was a boy aged five and one-third years (No. 561, 1895).

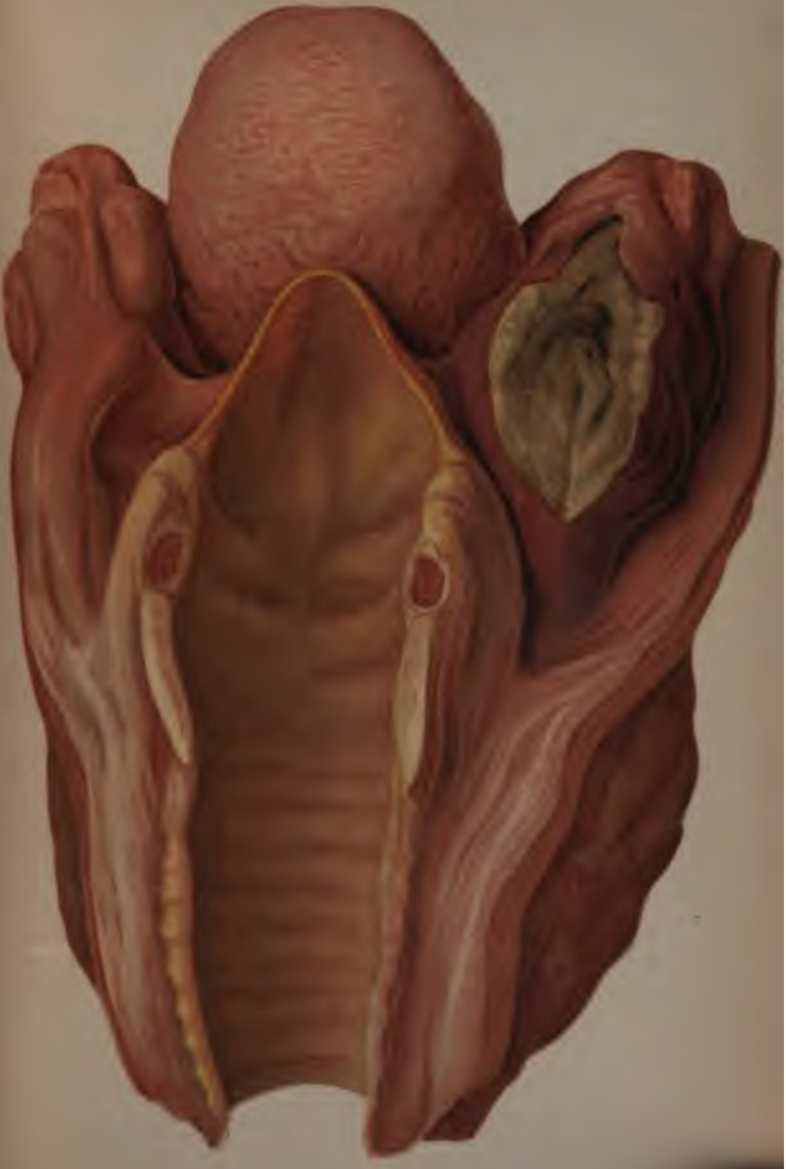
Explanation of Plate 34.

ABSCESS OF THE POSTERIOR AND LATERAL PHARYNGEAL WALL (PARA- AND RETRO-PHARYNGEAL ABSCESS).

Sanious abscess, the size of a large plum, developed without demonstrable cause from a submucous circumscribed phlegmonous inflammation. The abscess had caused a peracute septicæmia, *i.e.*, metastatic purulent fibrinous pleurisy of the left side, moderate swelling of the spleen, and parenchymatous hepatitis.

The patient was a man aged forty-two years (No. 424, 1895). Clinically and anatomically the condition was similar to angina Ludovici, in which the abscesses, cryptogenic in their origin, are met with in the region of the base of the tongue, the lower maxilla, and the submaxillary region.

Tab. 34



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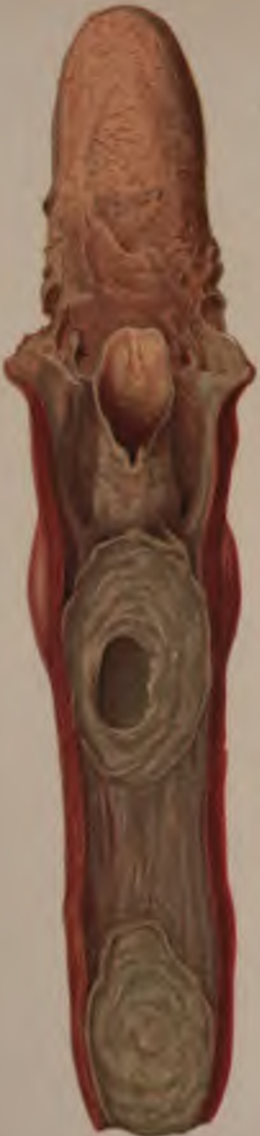

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Explanation of Plate 35.

Carcinoma of the Œsophagus, Perforation into the Trachea.

(Two-fifths natural size.)

Below the larynx is an annular and oval carcinomatous ulcer of a gray color; the bottom of the ulcer communicates with the lumen of the trachea by a large opening. Some distance farther down is a similar, somewhat smaller non-perforating ulcer. Death by gangrenous inhalation pneumonia of both lower lobes.

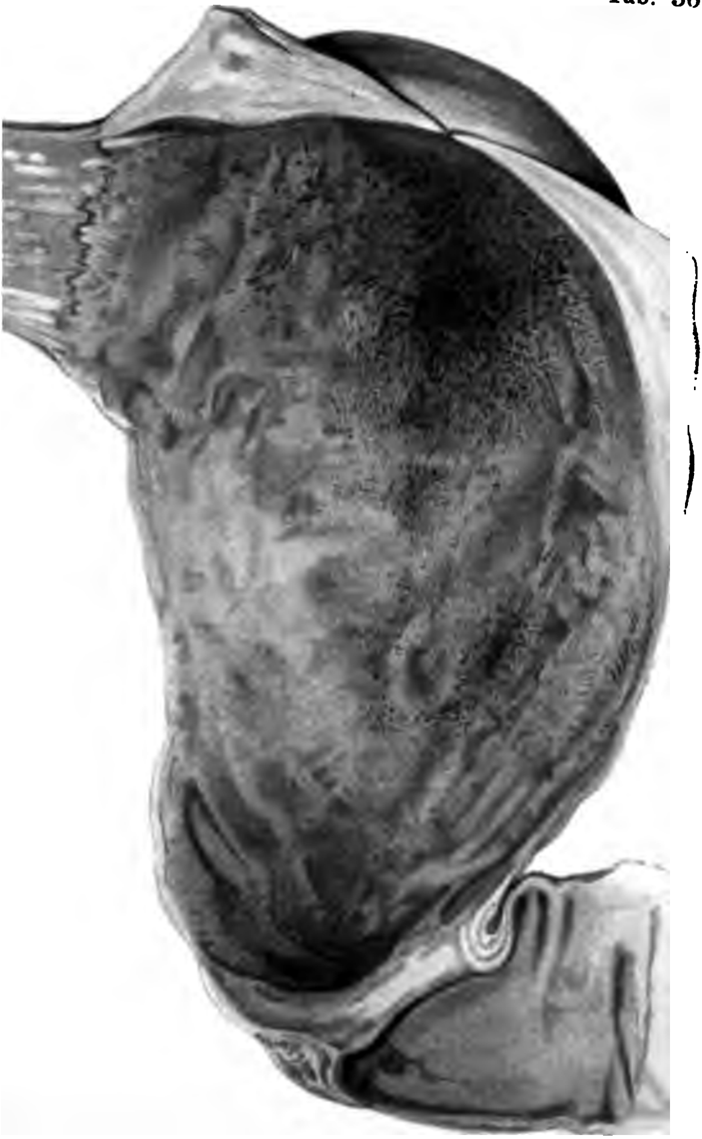
Explanation of Plate 36.

HYPOSTATIC HYPERÆMIA OF THE GASTRIC MUCOSA AT THE FUNDUS.

(Two-thirds natural size.)

The mucous membrane at the fundus and at the posterior wall is of a diffuse brownish-red color.

Tab. 36.



Lith.Anst.v.F.Reichhold, München

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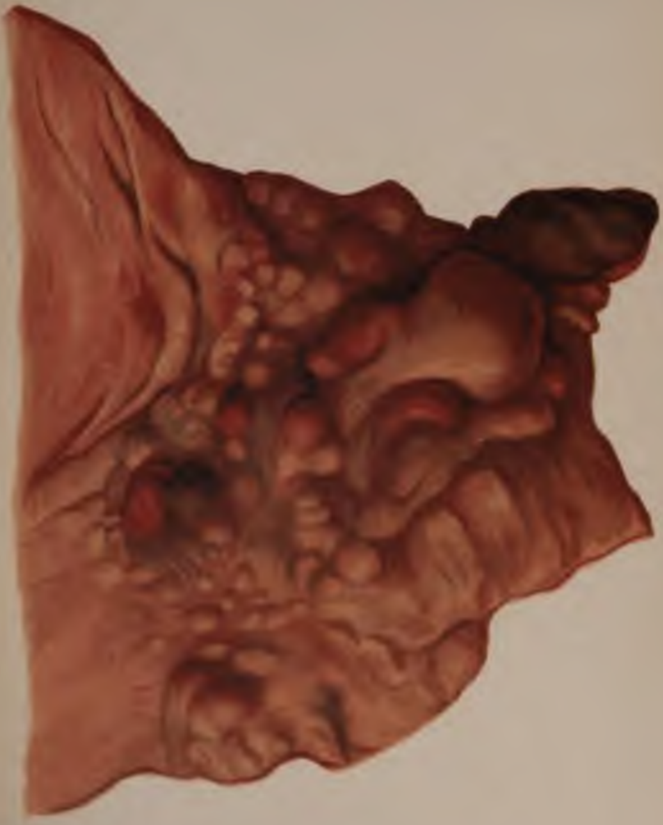


Lith.Anst.v. F. Reichhold, München.

Explanation of Plate 37.

MULTIPLE POLYPI OF THE MUCOUS MEMBRANE OF THE PYLORIC PORTION OF THE STOMACH.

The pyloric portion of the stomach, over a region the size of the palm of the hand, is covered with a large number of spherical or pear-shaped polypi; the smaller of these are rather flat, the size of a hempseed, the rest ranging from the size of a pea to that of a cherry; they are semisoft in consistence, in part bright red, and have a juicy lustre. The affected portion of the gastric mucosa is markedly thickened and hypertrophic—chronic gastritis polyposa.

Explanation of Plate 38.

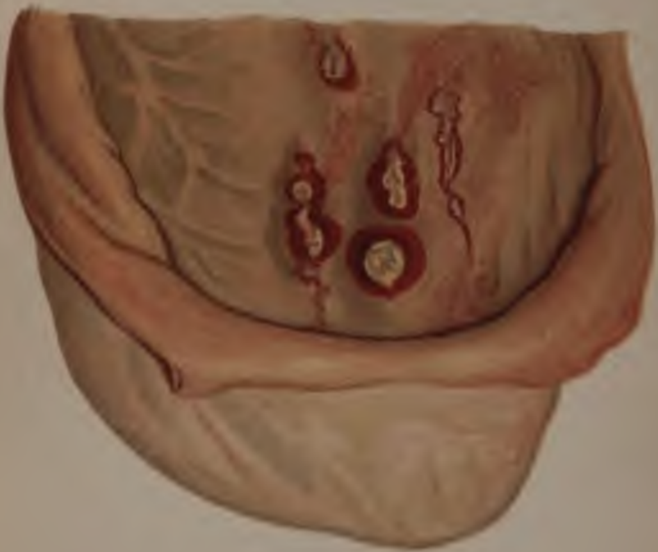
Tuberculous Ulcers of the Stomac

At the fundus of the stomach, in the slightly mucous membrane, are several roundish and dently rather recent ulcers with deep red ar bottom of the ulcers is of a dull yellowish-wh

The cause of death of the patient (a boy years, No. 274, 1895) was a chronic tubercul tonitis, with cheesy tuberculosis of the retro and mesenteric lymph glands, and confluent pneumonia of the left side; there was also pr general emaciation.

(The case is described at length in the disser Kanzow: "Ein Beitrag zur Casuistik der tub Magengeschwüre," Munich, 1895.)

Tab. 38.



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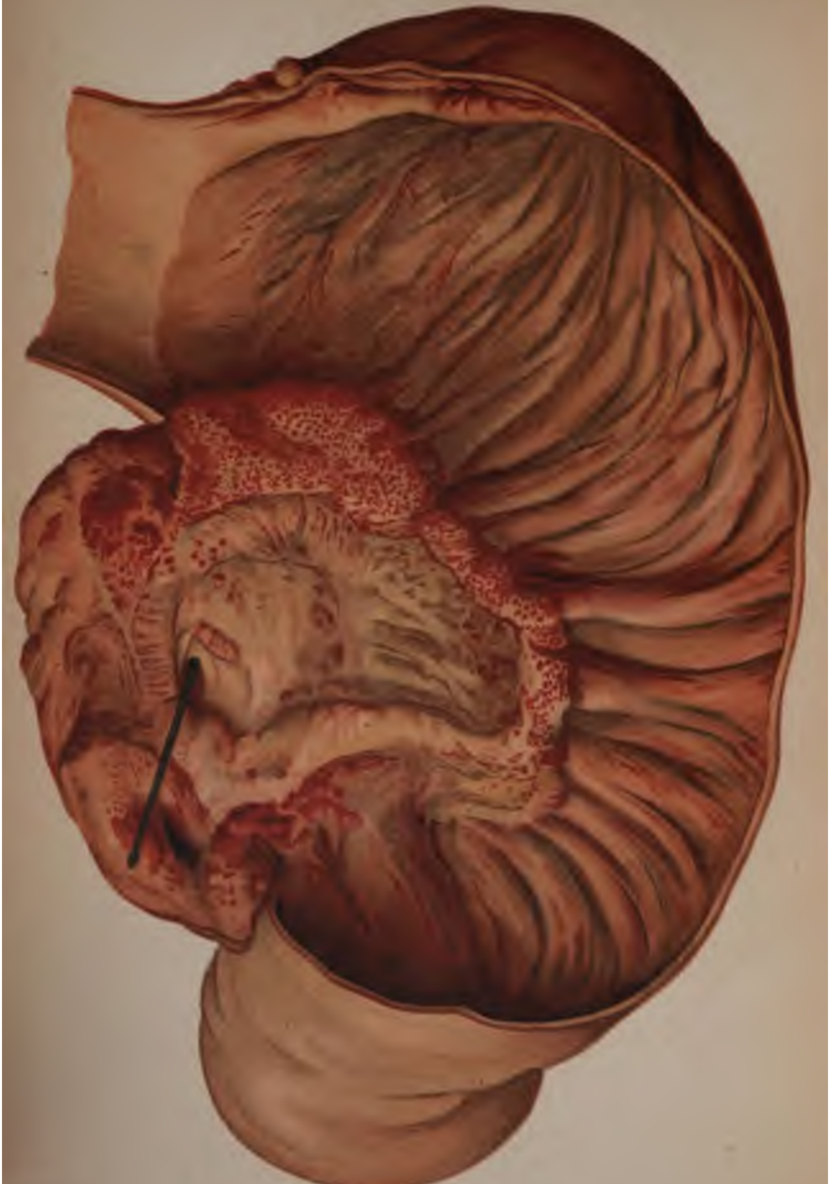

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Explanation of Plate 39.

COLLOID CANCER OF THE STOMACH.

The cardiac portion of the lesser curvature and the posterior wall of the stomach are occupied by a nearly oval tumor, the size of the palm of the hand, having a rather firm, wall-like, tumid margin; in its centre is an ulcer the size of a dollar. Corresponding to the upper portion of the ulcer the gastric wall is much thinned and translucent over an area the size of a dime. Within this thinned area is a perforation readily admitting a goose quill and opening into the abdominal cavity; the direction of the perforation is directly backward toward the spinal column. Both in the tumid margin and at the bottom of the ulcer we observe scattered through the cancerous mass semi-translucent colloid droplets, surrounded by a reticulated fibrous framework.

The external surface of the stomach is united to the anterior abdominal wall by a firm, indurated tissue (perigastritis adhæsiva); the region of the lesser curvature is greatly shortened and, corresponding to the tumor palpable from without, is covered with flat yellowish nodules, the largest the size of a lentil.

Cause of death—acute pyo-fibrinous perforation peritonitis. There were also extreme general emaciation and brown atrophy of the heart.

The patient was a man aged fifty-eight years (No. 136, 1895).

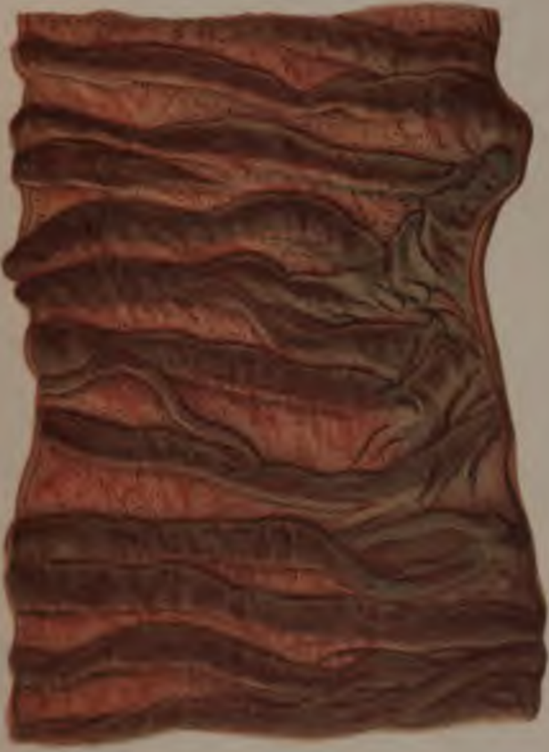
Explanation of Plate 40.

CHRONIC ENTERITIS PIGMENTOSA.

The mucous membrane of the jejunum appears much folded, swollen, succulent, reddened, and pigmented with slaty punctiform spots. The entire intestinal wall is friable like tinder and thickened; the contents of the bowel are watery and abundant.

The extremely emaciated patient (weight, 39 kgm. = 86 lb.), aged sixty-seven years, had died of chronic pulmonary tuberculosis (No. 172, 1895).

Tab. 40.



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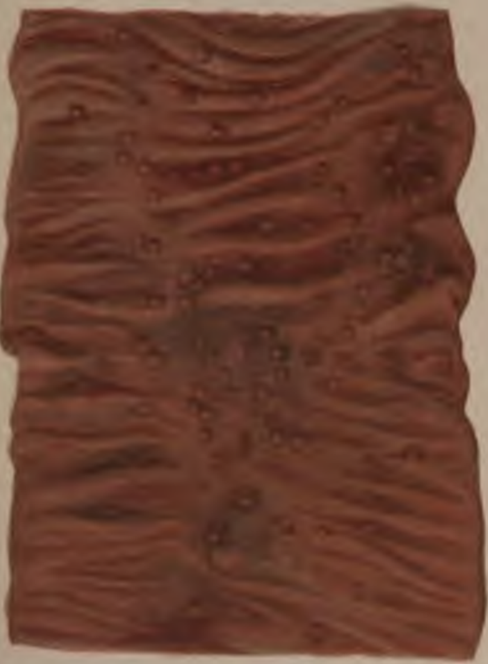


Lith.Anst.v. F. Reichhold, München.

Explanation of Plate 41.

CHRONIC FOLLICULAR COLITIS.

The mucous membrane shows a dull brownish-red discoloration and is swollen, the solitary follicles throughout are enlarged to a size from a pin's head to a hemp-seed. Throughout the small intestine the mucous membrane is more atrophic but otherwise similar, and the follicles are likewise much enlarged.

The patient was an extremely atrophic nursling, aged seven months, and the terminal affection was capillary bronchitis and beginning lobular pneumonia. General anæmia and atrophy of all organs were present. During life the symptoms were those of chronic gastro-enteritis and lobular pneumonia. Length of body, 58 cm. = 23 in.; weight, 2.6 kgm. = 5 lb. 11½ oz. (No. 710, 1895).

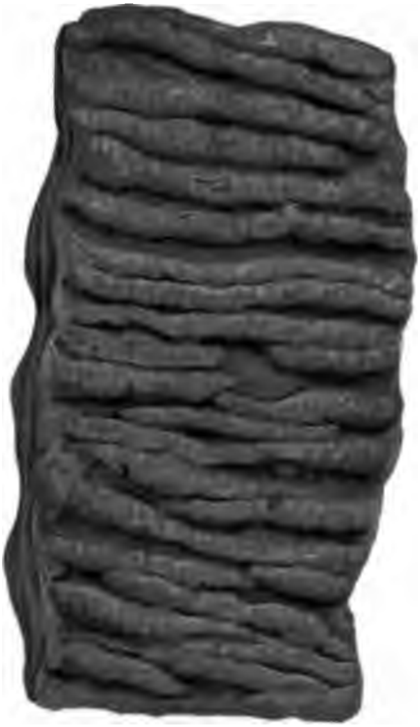
Explanation of Plate 42.

DIPHTHEROID (NECROTIC) ENTERITIS.

The wall of the small intestine is greatly thickened, of a faded brownish-red color, and very friable. The mucous membrane is markedly folded; the folds are widened, fluctuating, and very juicy. At the top of the folds the upper layers of the mucous membrane are furfuraceous and of a greenish-yellow color. The contents of the bowel are watery and offensive.

The patient, a woman aged twenty-eight years, was slender, extremely emaciated (weight, 30.5 kgm. = 67¼ lb.), and syphilitic at the same time. The cause of death was recurrent tuberculosis of the lungs associated with ulcerative tuberculosis of the colon. The cause of the serious intestinal disease could not be exactly determined, perhaps it was due to mercurial treatment (No. 520, 1895).

Tab. 42



Lith. Anst. v. F. Reichhold, Münch.

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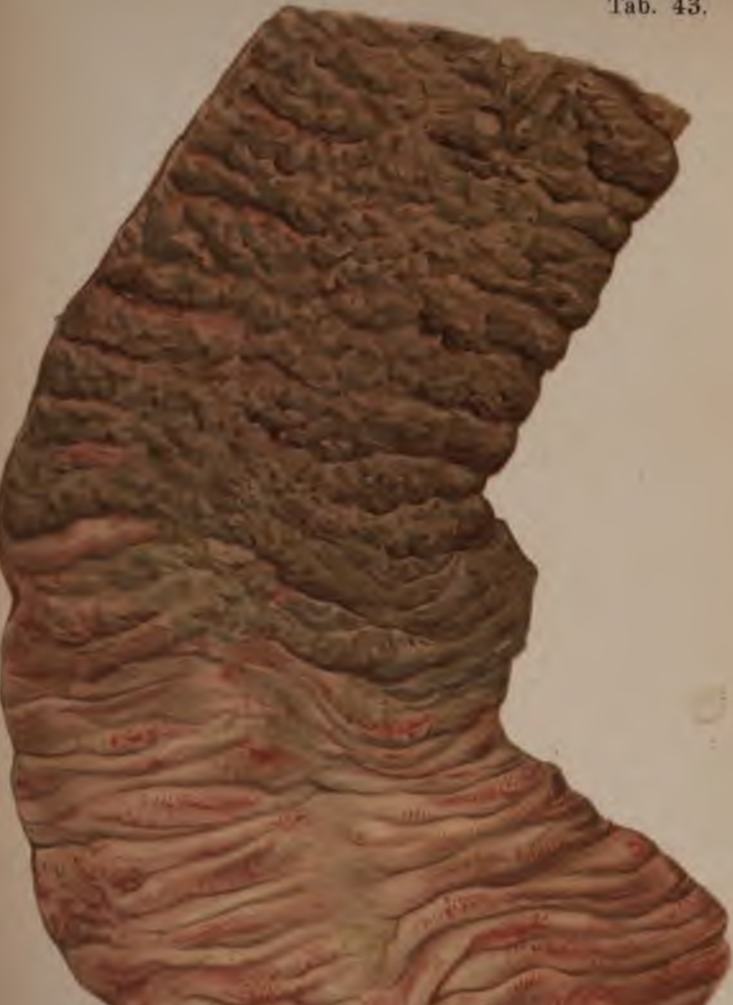

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Explanation of Plate 43.

TOXIC ENTERITIS (MERCURIAL DYSENTERY).

The mucous membrane in the upper portions of the rectum is deep red and covered at the top of the folds with discolored membranous masses.

The patient, aged twenty-five years, had died six days after delivery of puerperal sepsis and purulent phlegmons of the pelvic connective tissue. During attempts at forceps extraction outside of the clinic a perforating laceration of the left vaginal wall, 6 cm. = $2\frac{3}{8}$ in. long, had occurred. At the clinic the child was delivered with forceps and the laceration was sutured. Before the application of the forceps the vagina was irrigated with 0.1-per-cent sublimate solution, when the fluid evidently passed through the laceration into the pelvic cellular tissue. (For the illustration and notes of the case the author is indebted to Prof. v. Hofmann, of Vienna.)

Explanation of Plate 44.

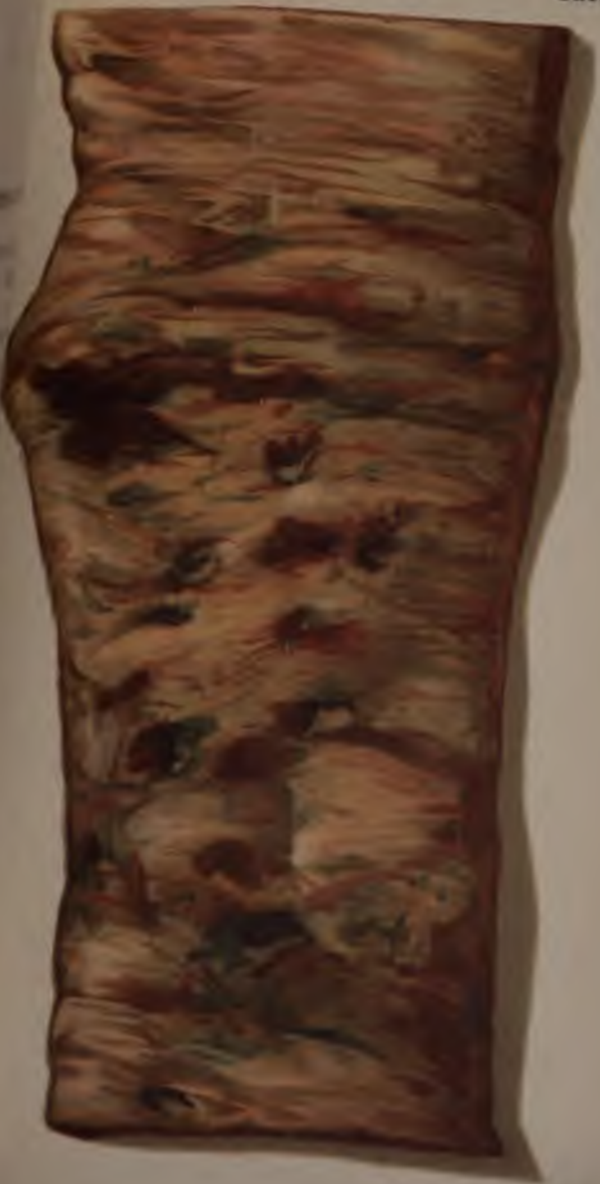
NECROTIC AND ULCERATING SEPTIC COLITIS. SPORADIC DYSENTERY.

The mucous membrane over the entire extent of the colon is greatly injected, œdematous, with superficial necrotic spots here and there. At various points are circumscribed, roundish, ulcerated defects, extending in some instances as far as the serosa, and having a yellowish discolored, soft, and friable bottom; there are also multiple hemorrhagic foci of the mucosa.

The patient, a woman aged seventy years, was insane and marasmic. The cause of death was a terminal pleuropneumonia of the left lower lobe. She had been bedridden for months and had long suffered from obstinate constipation. In the last weeks before death profuse diarrhœas and symptoms of an intense chronic catarrh of the colon set in. As a large bedsore in the sacral region was present between three and four weeks before death, it is probable that the grave intestinal affection was of septic origin (No. 165, 1894).

(The case is described at greater length in the dissertation by Maximo Asenjo: "Zur Kenntniss der sporadischen Dysenterie," Munich, 1894.)

Tab. 44.



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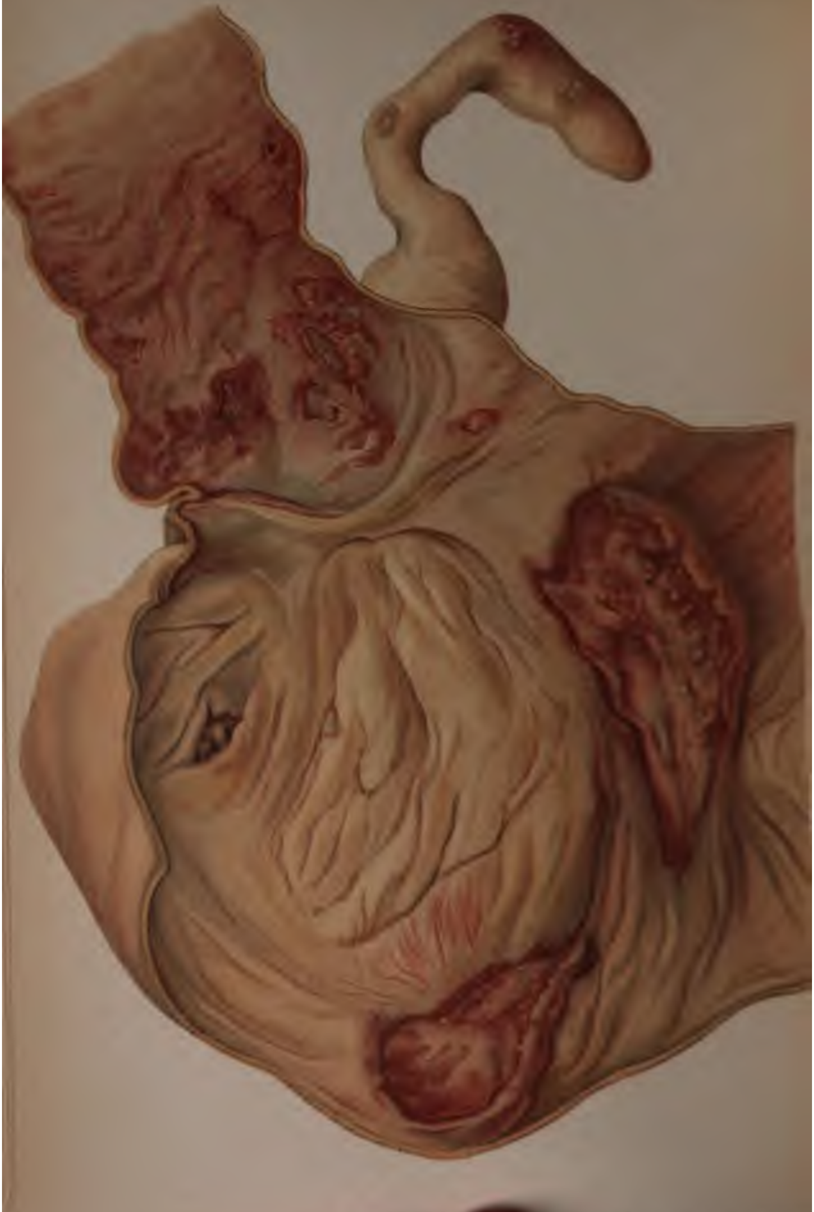


Lith.Anst.v.F.Reichhold, Münch

Explanation of Plate 45.

TUBERCULOUS ULCERS OF THE SMALL AND LARGE INTESTINE.

In the lower portion of the ileum, immediately above the valve of Bauhin, is a group of irregular ulcers with elevated red margins; in the latter and at the bottom of the ulcers yellowish miliary tubercles are visible. At the point where the cæcum passes into the ascending colon are two larger, transverse ulcers similar to the others—with red, tumid, and partly undermined margins. At the bottom of the ulcers is a larger number of miliary, yellowish-white nodules. Upon the injected serosa of the thickened appendix vermiformis are groups of secondary miliary tubercles; each group corresponds to a tuberculous ulcer in the interior of the appendix.

The ulcerating intestinal tuberculosis was a secondary condition, the primary lesion being a fatal chronic pulmonary tuberculosis in a patient aged twenty-six years (No. 74, 1895).

Explanation of Plate 46.

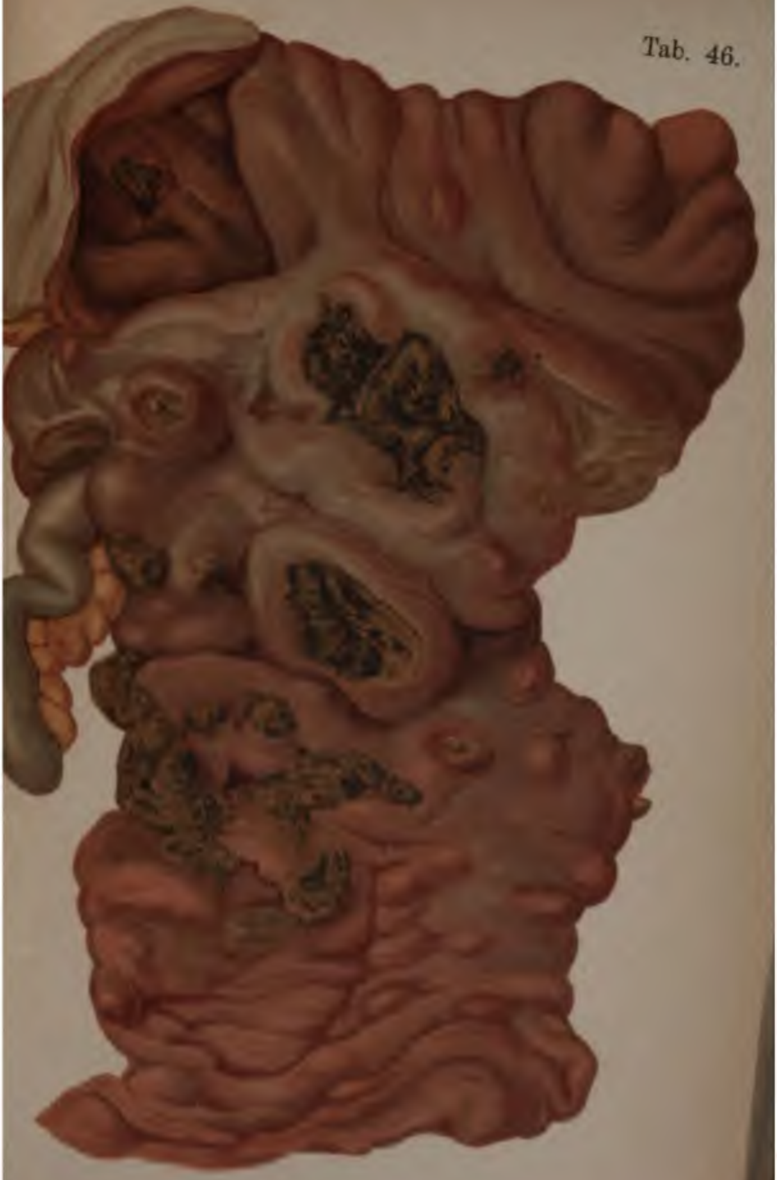
TYPHOID FEVER.

(End of the second week.)

In the greatly injected and swollen mucosa of the lower ileum above the valve of Bauhin is a considerable number of ulcers covered with firm crusts showing a dirty-greenish discoloration. Two larger oval ulcers with markedly tumid margins are situated immediately above the valve. In the neighborhood are several cup-shaped ulcers, ranging in size from a lentil to a pea, which are likewise covered with crusts. While the larger ulcers correspond to Peyer's patches, the smaller spring from the solitary follicles which are swollen to the size of a pea; some of these, in the stage of medullary swelling and infiltration (lymphoid, inflammatory hyperplasia), are clearly differentiated from the injected and swollen mucosa in the lower portion of the illustration. In the adjoining cæcum the mucosa resembles that of the ileum; it is markedly swollen, injected, and shows an enlarged solitary follicle and toward the left a small ulcer covered with a crust.

The autopsy of the patient, a woman aged twenty-seven years (No. 619, 1895), revealed medullary swelling of the mesenteric glands, enlargement of the spleen (weight, 410 gm. = 14+ oz.), and a hemorrhagic [illegible] of the right lower lobe.

Tab. 46.



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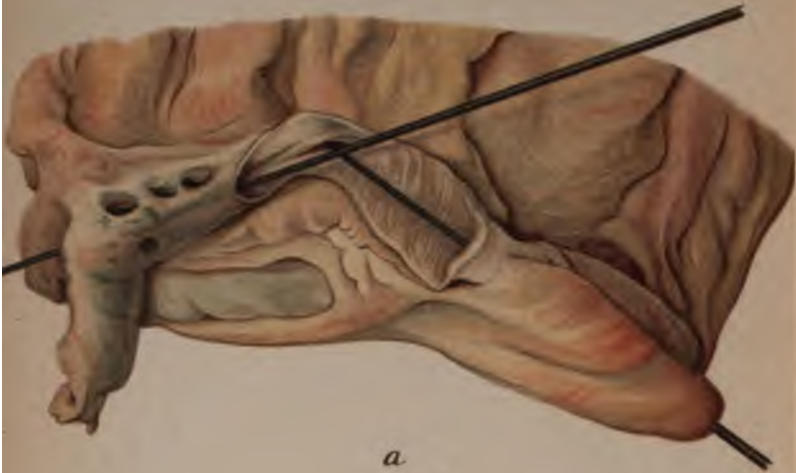

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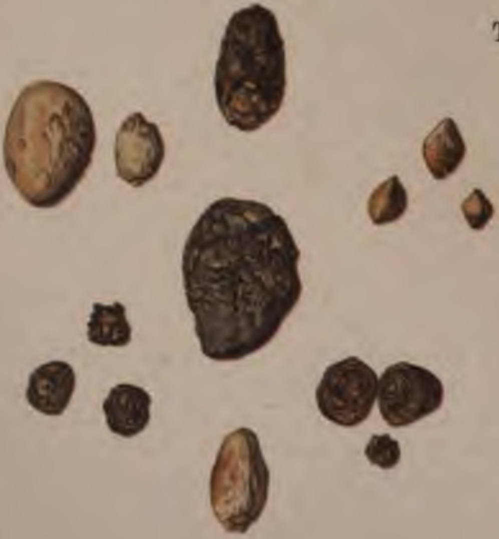

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Explanation of Plate 47.

ULCERATIVE AND PERFORATING APPENDICITIS.

FIG. *a*.—In the peripheral portion of the appendix vermiformis are several perforations caused by fæcal concretions. The perforation was followed by a purulent retroperitoneal paratyphlitis and the formation of a large abscess which, like a psoas abscess, burrowed from the lower margin of the right kidney under Poupart's ligament and into the true pelvis. The course was subacute. Fatal issue with symptoms of exhaustion.

The patient, a woman aged eighteen years (No. 287, 1895), was found at the autopsy to be greatly emaciated and extremely anæmic.

(Further details about this case will be found in the dissertation by Franz Deutschländer: "Ueber Appendicitis perforativa mit sekundärer eiteriger retroperitonealer Paratyphlitis," Munich, 1895.)

FECAL CONCRETIONS FROM THE VERMIFORM APPENDIX.

(Seven cases.)

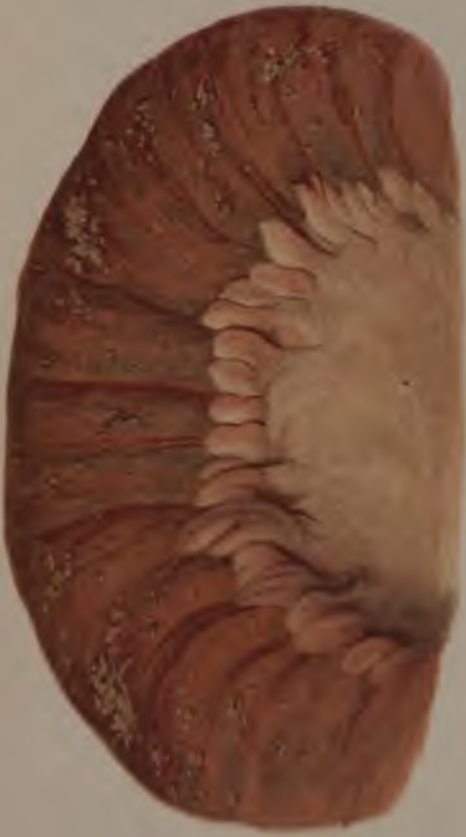
FIG. *b*.—Three separate concretions, from the size of a bean to that of a cherry (in the centre of the figure), and four groups of such at the periphery. The concretions differ considerably in color and bulk; on section (to the right below) they are distinctly laminated.

All of these concretions from seven cases gave rise to ulcerating necrotic appendicitis and fatal perforation peritonitis. The patients (three men, four women) were between twelve and thirty-eight years old; the average age was twenty-two years.

Explanation of Plate 48.

METASTATIC TUBERCULOSIS OF THE PERITONEUM.

Numerous miliary, grayish-white tubercles, mostly arranged in groups, are situated on the slightly injected serosa of the small intestine. Each group corresponds to a tuberculous ulcer located on the mucous surface. Besides, disseminated miliary tubercles in moderate numbers are scattered over the serous covering of the intestine, somewhat as in miliary tuberculosis of the entire peritoneum.



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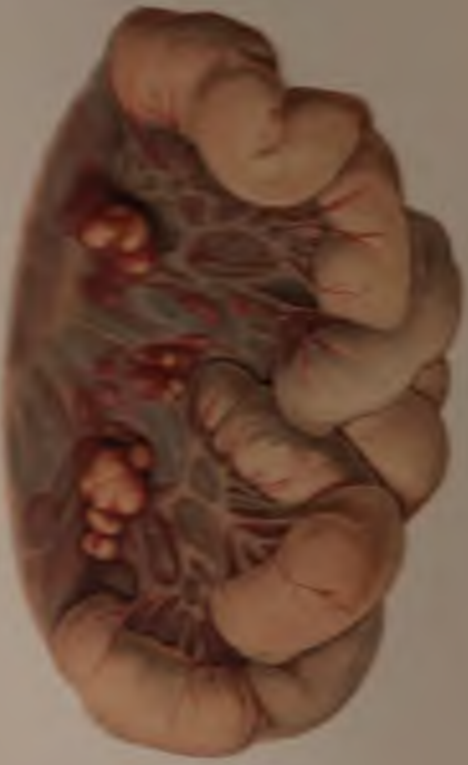

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Explanation of Plate 49.

CHEESY TUBERCULOSIS OF THE MESENTERIC LYMPHATIC GLANDS.*

In the mesentery are found a larger number of swollen lymphatic glands, ranging in size from a hempseed to a cherry, which are distinctly differentiated from the surrounding structures by their yellowish-white color and their partially somewhat reddened capsules. The cut surface of the glands resembles in color and consistence that of a raw potato.

The specimen was derived from a child, aged four months (No. 379, 1895), which had died of an acute general miliary tuberculosis of the lungs, liver, and spleen, which was secondary to an older cheesy tuberculosis of the lymphatic glands of the neck and of the cavities of the thorax and abdomen. The associated conditions were caries of one petrous bone, furunculosis, intestinal catarrh, and general atrophy. The child's mother had died soon after the delivery of tuberculosis with extremely rapid course. (Infection of the child by the mother early after its birth.)

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Explanation of Plate 50.

SUBCHRONIC AND SUBACUTE TUBERCULOSIS OF THE PERITONEUM.

The peritoneal investment of the intestine and mesentery is injected here and there and shows some inflammatory thickening. Situated upon it are numerous nodules, ranging in size from a pin's head to a pea or bean, yellowish-white in color and partly somewhat flattened; some of them are composed of an agglomeration of several smaller ones. The nodules bear a close resemblance to metastatic cancerous proliferations.

The patient, aged fifty-four years, suffered from a primary genital tuberculosis of the uterus and tubes, so that the peritoneal tuberculosis can be almost certainly ascribed to the disease of the Fallopian tubes. There were also found tuberculous ulcers in the colon and a local tuberculosis of the left lung (No. 383, 1895).

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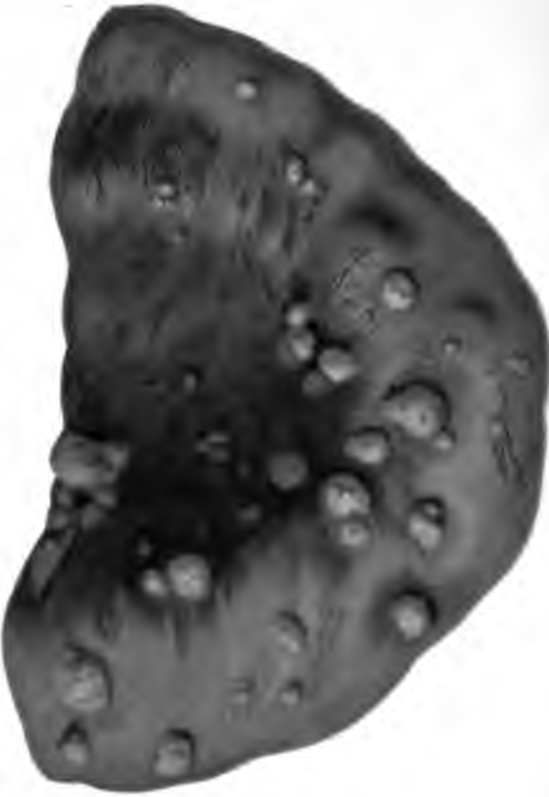

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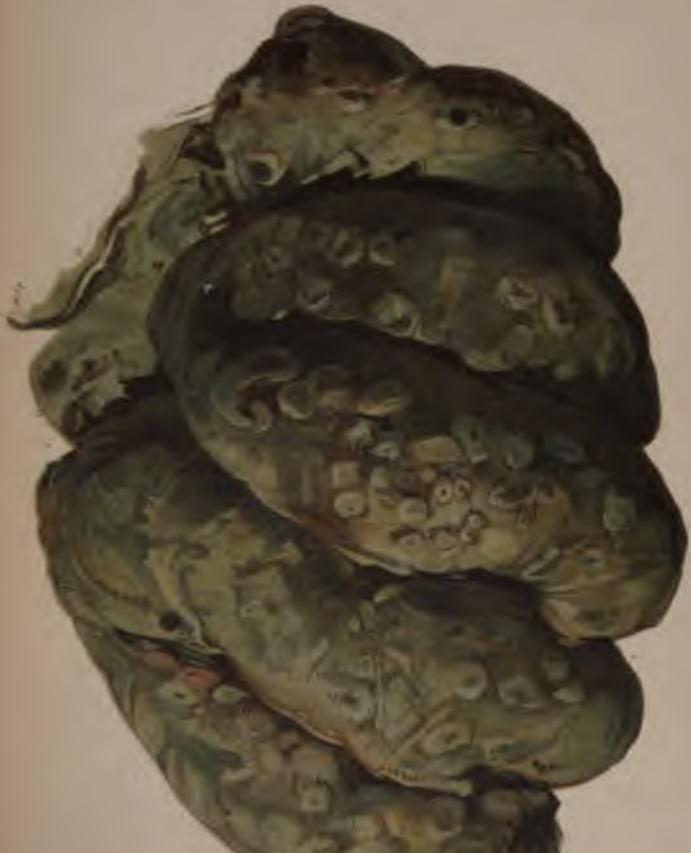

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Explanation of Plate 51.

Carcinosis of the Peritoneum.

Upon the peritoneum which shows a greenish discoloration* are numerous flat nodules of a whitish color, the majority the size of a lentil or pea. The abdominal cavity contained about 1½ litres (quarts) of a serous amber-colored liquid (ascites). The omentum (not visible in the plate) showed a marked cancerous infiltration, forming a misshapen, tumid, and lobulated mass with diffuse bloody infiltration and covered with numerous whitish nodules.

The location of the primary carcinoma of the patient, aged forty-three years (No. 190, 1894), was in the ovaries. The layers of the pleura presented a similar appearance to that in the abdominal cavity—disseminated cancerous nodules with hemorrhagic exudation. Associated conditions noted were chronic parenchymatous nephritis, fatty liver, and anasarca of the lower extremities.

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* The intense greenish discoloration of the peritoneum became more pronounced after death.

Explanation of Plate 52.

FATTY AND ENGORGED LIVER IN CHRONIC PULMONARY TUBERCULOSIS.

FIG. *a.*—The liver is enlarged, of a pale orange-red color, and diminished consistence. The contained blood is very small in amount. Light scraping produces a viscid juice with fatty lustre. The acinous marking is very distinct; the centre of the acini is light brown, the marginal portions are pale yellowish in color. The microscopical appearance is that of chronic fatty infiltration, with ectasia of the capillaries in the centre of the acini, *i.e.*, the organ is the anæmic engorged liver with fatty infiltration, which is observed with particular frequency in connection with chronic pulmonary tuberculosis.

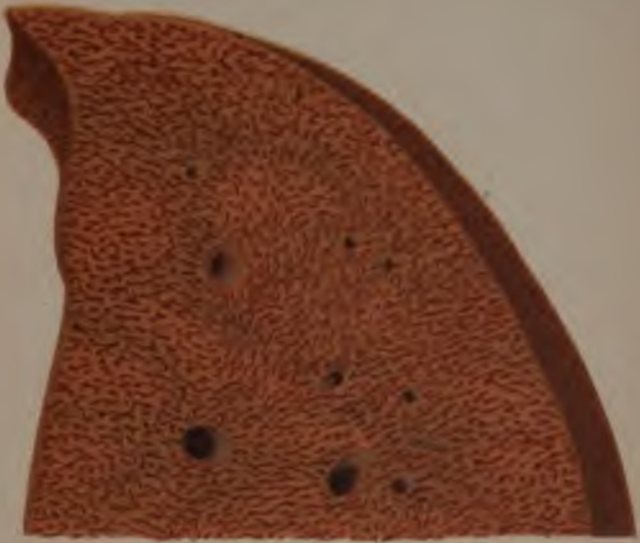
The patient (No. 633, 1894), aged twenty-five years, had succumbed to chronic and recurrent tuberculosis of the lungs with pneumothorax and ulcerating intestinal tuberculosis.

FATTY INFILTRATED NUTMEG LIVER. CHRONIC ENGORGEMENT OF THE LIVER IN HEART DISEASE.

FIG. *b.*—The liver is somewhat reduced in size and harder to cut than the normal organ. The cut surface shows the nutmeg marking very clearly and is decidedly icteric. The centres of the lobules are dark brown, the marginal portions dull yellowish in color. The contained blood is diminished in amount, probably by reason of prolonged stay in bed of the patient.

The patient, a woman aged fifty-four years (No. 47, 1895), had suffered from chronic aortic sclerosis, extension of the inflammatory process to the aortic valves, with consequent insufficiency and stenosis.

Tab. 52.



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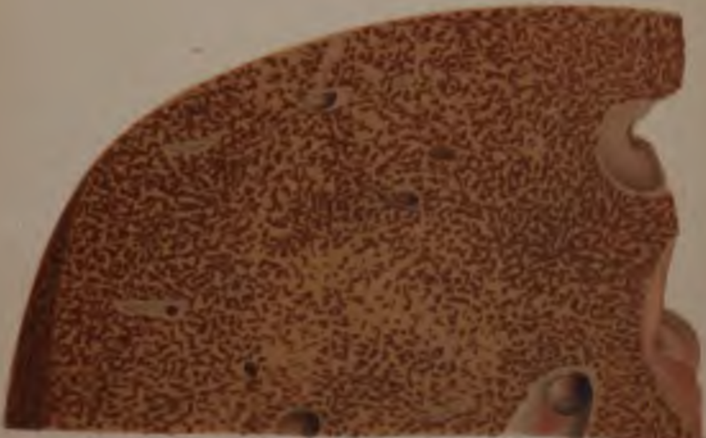

b

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Tab. 53.

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Explanation of Plate 53.

CHRONIC JAUNDICE (ICTERUS VIRIDIS) WITH ADENOCARCINOMA OF THE LIVER.

The liver is enlarged, the surface somewhat uneven, and of a dark yellowish-brown color. Hardly any blood escapes from the cut surface, but a profuse quantity of bile instead. The larger bile ducts are considerably dilated and filled with bile. The parenchyma of the liver is of a dark olive-green color (icterus viridis).

The cause of the mechanical icterus was found at the porta of the liver, in the shape of a carcinoma which had proliferated chiefly into the hepatic duct and thus had made the escape of the bile impossible. Duration of the icterus in the patient, aged sixty-five years (No. 57, 1895), about three and a half months.

Explanation of Plate 54.

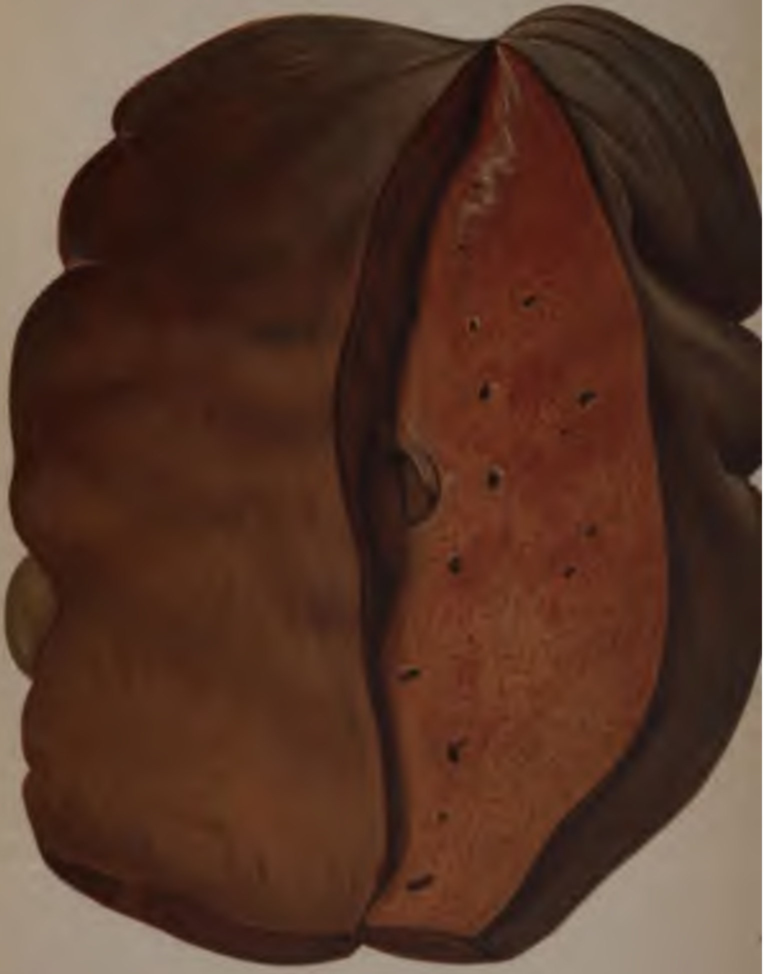
ACUTE YELLOW ATROPHY OF THE LIVER.

(Two-thirds natural size.)

The liver is reduced in all its diameters, altogether to nearly one-half of its normal bulk (weight, 920 gm. = 2 lb.). The capsule is wrinkled; the consistence is tough, very relaxed, and limp. The cut surface is partly icteric, partly diffusely reddish in color. The acinous marking is completely obliterated. Blood contents very scanty. On light scraping we obtain a yellowish colored, turbid juice in considerable amount.

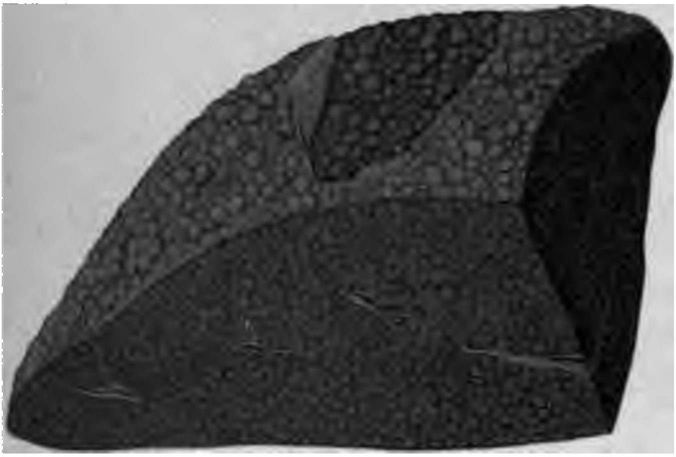
Other conditions found in the patient, aged twenty-one years (No. 161, 1895), were marked general icterus, fatty metamorphosis of the heart and kidneys, multiple subserous ecchymoses of the pleura, peritoneum, epicardium, and endocardium.

Tab. 54.



Lith. Anst. v. F. Reichhold, München.

Tab. 55.



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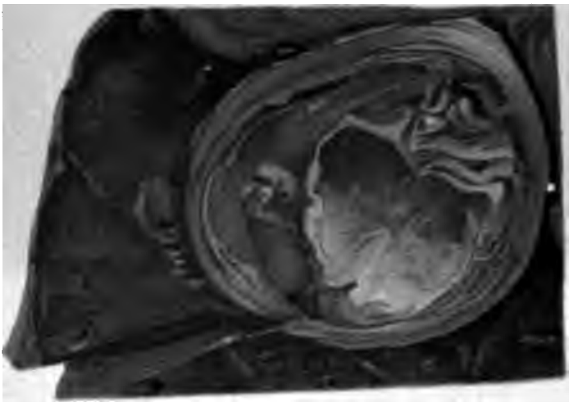

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Lith.Anst. v. F. Reichhold. München

Explanation of Plate 55.

ATROPHIC CIRRHOSIS OF THE LIVER.

FIG. *a.*—The liver is considerably reduced in size; the surface is coarsely granular, pale grayish-yellow in color. On section the creaking is distinctly heard; the parenchyma is almost bloodless, dry; the cut surface is likewise uneven and rough. Between the irregularly shrunken lobules the interacinous connective tissue is seen to be greatly though also irregularly developed.

The patient, aged thirty-five years (No. 70, 1895), was affected with an angiofibroma of the sacral region; death by hemorrhage from the neoplasm.

ECHINOCOCCUS CYST OF THE LIVER (PARASITE DEAD).

FIG. *b.*—A cyst nearly as large as a hen's egg is present in the parenchyma of the liver. The fibrous capsule is thickened. Within is the collapsed, laminated cyst of chitin, partly detached from the wall, here and there still milky in color.

Other conditions found in the patient, aged fifty-eight years (No. 278, 1894), were aortic sclerosis, secondary insufficiency of the aortic valves, granular atrophy of the kidneys, and a terminal purulent pleurisy over the right lower lobe.

Explanation of Plate 56.

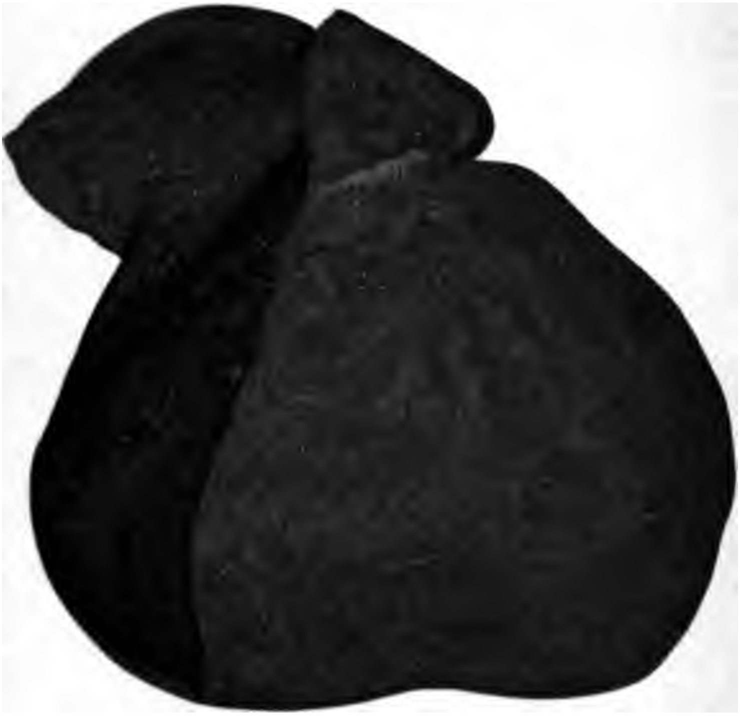
ACUTE MILIARY TUBERCULOSIS OF THE LIVER.

(Natural size.)

Scattered through the dark brownish-red liver tissue are numerous, minute, punctiform nodules of a gray and yellowish-gray color, most of them opaque yellowish in the centre.

In the patient, a boy aged five years (No. 615, 1894), the spleen and kidneys were found similarly sprinkled with miliary nodules. Other conditions found were an older cheesy tuberculosis of the peribronchial and mesenteric lymphatic glands, cheesy lobular pneumonia, tuberculous pleurisy, and sero-fibrinous pericarditis. The exacerbation of the tuberculous process was observed in connection with whooping-cough.

Tab 56.



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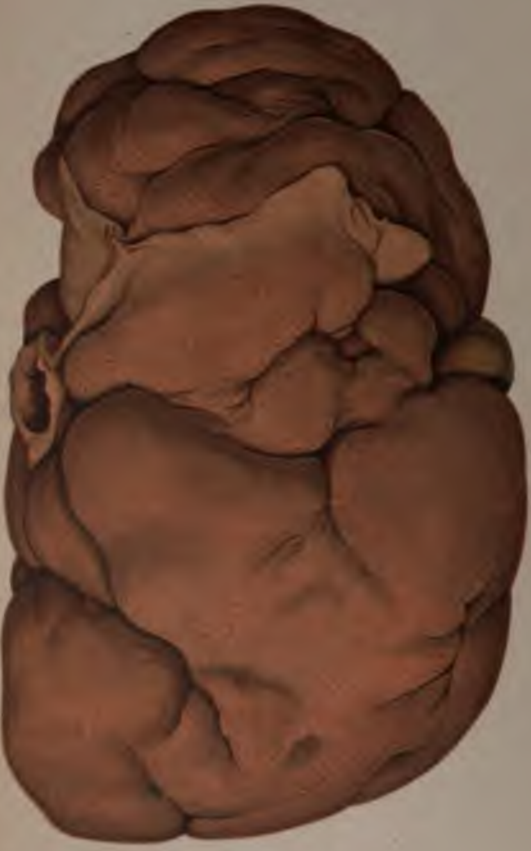
Tab. 57.



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Explanation of Plate 57.

SYPHILIS OF THE LIVER. LARGE-LOBED LIVER.

(Two-thirds natural size.)

The entire liver is reduced in size and very much deformed, being divided into irregular lobes by numerous, deep, cicatricial retractions. The sharp margins are rounded; the tissue is firm and tough in consistence. On the cut surface we observe, corresponding to the cicatricial retractions, firm connective-tissue condensations.

An old specimen from the collection.

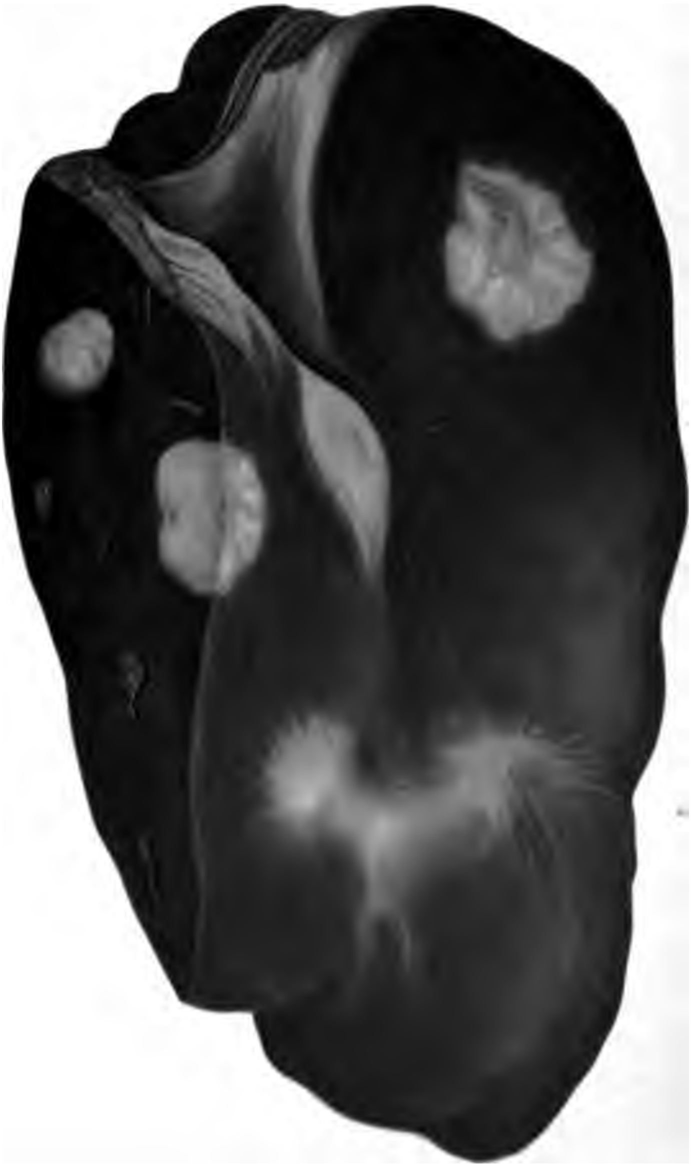
Explanation of Plate 58.

SYPHILIS OF THE LIVER. MULTIPLE GUMMATA.

The liver is somewhat reduced in size, full of blood, and the acinous markings are obliterated. In the parenchyma, partly situated subperitoneally, are three roundish, yellowish-white foci of firm consistence, ranging in size from a cherry to a hazelnut, *i.e.*, syphilomata. The milky opacity of the capsule over the lower portions of the liver corresponds to a shallow furrow caused by tight lacing.

The greatly emaciated patient, a woman aged thirty-four years (No. 600, 1895), was syphilitic, having specific ulcers upon the labia and in the vagina. Other conditions found were defect of the nasal bones (depressed nose), amyloid spleen, and progressive tuberculosis of both upper lobes.

Tab. 58.



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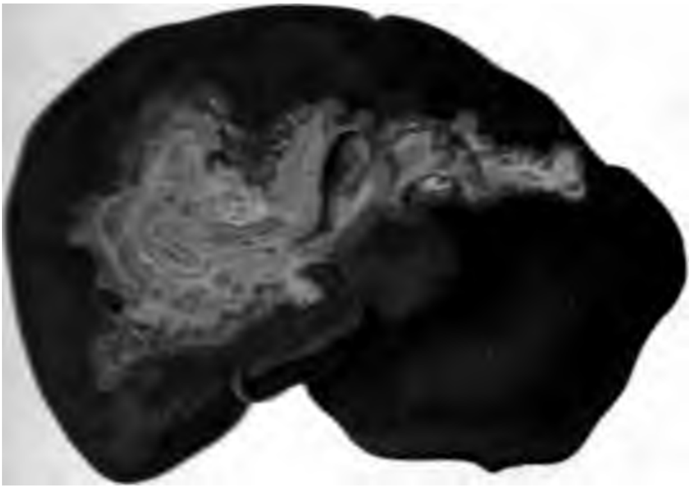
Tab. 59.



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Explanation of Plate 59.

SYPHILIS OF THE LIVER IN THE NEW-BORN.

Compared with the development of the body the liver appears enlarged; weight, 144 gm. = 5 oz.; consistence very firm. On section we observe, surrounded by a light yellow, partly sulphur-colored and icteric parenchyma, a syphiloma the size of a large plum, having an irregularly oblong form, prolongations of which extend through all the lobes; it is of a firm, almost board-like consistence, of a yellowish-gray color, with irregular jagged borders which are somewhat overhanging; in part it is gelatinous and sprinkled with grayish-yellow miliary nodules. Associated conditions found were general icterus, great swelling of the spleen to three times its size, congenital syphilis.

The illegitimate child, born of a syphilitic mother, had lived twelve hours; length of the body, 42 cm. = 16⅘ in.; weight, 1,500 gm. = 3 lb. 5 oz. (No. 376, 1894).

Explanation of Plate 60.

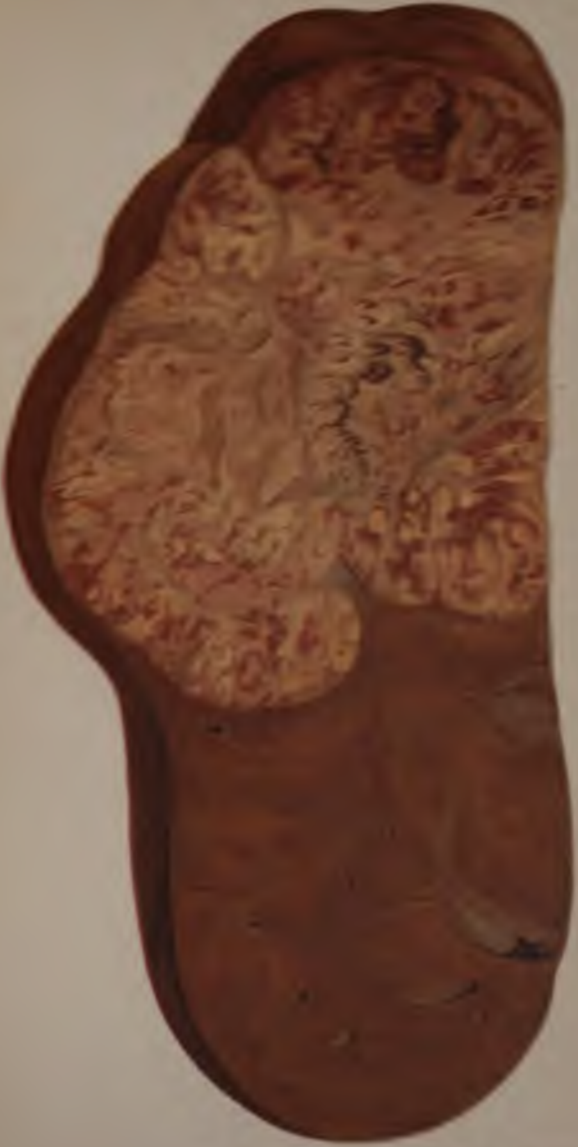
Primary Cancer (Medullary Carcinoma) of the Liver.

(One-half natural size.)

In the right lobe of the liver, which is nearly twice its normal bulk, is a tumor the size of an adult head, of a yellowish-white and reddish color, and of a medullary consistence. The cut surface is somewhat uneven; a few portions of the tumor have become softened, and these masses are mixed with dark blood. The gall-bladder is merged with the tumor.

In the greatly emaciated patient, a woman aged fifty-one years (No. 619, 1894), metastatic cancerous nodules were found in the brain (pons, oculomotor nucleus, and chiasm), lungs, heart, peritoneum, and left ovary.

Tab. 60.



Lith.Anst.v.F.Reichhold, Münc

PATHOLOGICAL ANATOMY.

Circulatory Apparatus, Spleen, Lymphatic Glands, and Thyroid Gland.

DISEASES OF THE PERICARDIUM.

THE diseases of the pericardium resemble in their etiological and anatomical relations those of serous membranes in general; only the tendency to tuberculous affections is less than that of the pleura and the peritoneum.

By far the most important anomaly of the pericardium is inflammation, namely:

PERICARDITIS.

(Plate 1.)

A distinction is to be made between the acute exudative forms and the chronic or terminated adhesive forms with partial or total obliteration of the pericardium.

Beside sero-fibrinous and hemorrhagic pericarditis we may mention purulent pericarditis, which is rare, and finallly tuberculous inflammation of the pericardium.

Aside from the traumatic form, idiopathic pericarditis is very rarely observed; more frequently its origin is hæmatogenous (articular rheumatism) and metastatic (in septico-pyæmia) or secondary by extension from surrounding structures; thus, cardial pericarditis is caused by the heart and its valves (endo-myo-pericarditis), and other inflammations are due to the pleura, the intrathoracic lymphatic glands, and the œsophagus.

Circumscribed forms have a strong tendency to remain stationary, to cause partial adhesion of the two layers of the pericardium, or to induce local thickenings and opacities of the pericardium (milk spots).

A benign form of pericarditis, tending to recovery and adhesion of the layers of the pericardium, occurs occasionally with acute rheumatic polyarthritis, also with pneumonia, pleurisy, dyscrasic conditions, chronic nephritis, chronic alcoholism, and tuberculosis; it is much more frequent in men than in women.

At first it is usually circumscribed; there are reddening, injection, with or without slight hemorrhages; the serosa presents a dull appearance, becomes opaque and velvety; a dull gray or pale reddish veil, resembling crape, covers the inflamed portions. The process extends from the visceral to the parietal layer, or *vice versa*. The fluid contents of the pericardium increase and become somewhat turbid. In the further course either absorption takes place, or, when the inflammation advances, a coherent pseudo-membrane forms, which is easily detached, has a whitish-gray color, and consists chiefly of fibrin.

When there is a steady increase of the fluid exudate and of the fibrinous products which cover the two layers of the pericardium more or less uniformly, the superficial strata of the coagulated fibrinous exudate present, by reason of the continuous contact and friction caused by the heart's action, a honeycomb appearance, a partly reticulated, partly villous arrangement (shaggy heart, cor villosum); while the deeper strata of the exudate soon give evidence of beginning organization, *i.e.*, an increased deposition of leucocytes, and the development of newly formed blood-vessels which sprout without cessation from the vessels of the inflamed serosa.

In the above-described sero-fibrinous inflammation the exudate is very frequently bloody (pericarditis hæmorrhagica), in which case the dilated pericardium often contains considerable quantities of a sero-sanguinolent fluid (as much as one to one and a half litres [quarts]) of a blackish-brown color.

Hemorrhagic pericarditis, which has a very unfavorable prognosis, runs an acute or subacute course, and is met with especially in drinkers (hence in men it is four times more frequent than in women and occurs oftenest between the fortieth and sixtieth year of life), in chronic nephritis, in cardiac hypertrophy (plethora), in cachectic and debilitated subjects affected with tuberculosis (two-thirds of all cases) or cancer.

In exudative cellulo-fibrinous pericarditis the affected layers of the pericardium present about the conditions of wound surfaces, that is to say, the embryonal granulation tissue interspersed with masses of fibrin gradually develops into mature connective tissue. All stages of this form may terminate in recovery, whose results may be observed at autopsies in all possible gradations, from simple circumscribed milky opacity and thickening of the pericardium (milk spots) to partial adhesion or total agglutination of both layers of the pericardium (partial or total adhesive pericarditis). In this way the exudative has changed to a productive inflammation, the liquid and amorphous solid products becoming absorbed, and in their place develops by way of substitution a vascularized fibrillary connective tissue which at first is rich in cells but later contains only few vessels and cells. In partial adhesive pericarditis fibrous connective-tissue trabeculæ (synechiæ) connect the two layers, while in total obliteration of the pericardium we find delicate or firm, sometimes membranous and calcified thickenings of the serous layers. In many cases of tardy or defective absorption the fibrinous exudate becomes thickened, caseous, and calcified; in the latter case callous and chalky masses surround like a shell smaller or larger portions of the heart muscle (pericarditis calculosa). That in the more intense forms of pericarditis the subepicardial external layers of the heart muscle may participate anatomically and functionally in the inflammatory process (peri-myocarditis) is easily understood.

Infectious pericarditis is chiefly represented in the purulent and tuberculous forms of inflammation.

PURULENT PERICARDITIS.

This form arises either through trauma or by continuity from surrounding structures; for instance, from infectious endocarditis and myocarditis, pleurisy, morbid processes having their primary seat in the mediastinum, the root of the lung, or in the intrathoracic lymphatic glands; or else from infectious and ulcerating processes of the lungs, the œsophagus, and more rarely the peritoneum. Lastly, a purulent pericarditis may be of hæmatogenous and metastatic origin in general sepsis. Owing to the multiplicity of the causative factors, the demonstration of the starting-point often requires particular care at the autopsy.

The first manifestations of purulent and fibrino-purulent inflammation coincide largely with the alterations present in sero-fibrinous pericarditis; in very debilitated anæmic subjects, in whom purulent pericarditis occurs occasionally as a terminal phenomenon, we find now and then no reddening and no fibrinous deposits on the layers of the pericardium; only the serous contents of the sac are somewhat increased, slightly turbid, and mixed with pus corpuscles in moderate quantities (sero-purulent pericarditis).

In other cases the exudate is abundant and fibrino-purulent; both layers are covered with a yellowish-white, incoherent, and lightly adhering pseudo-membrane. In the fibrinous masses numerous leucocytes are embedded, the free exudate is creamy and mixed with flakes of fibrin. The termination is usually fatal. When the course is more chronic we find, besides the liquid and partly coagulated exudate, that the layers of the pericardium are covered with a firm, coherent connective-tissue membrane, sometimes several millimetres thick, whose surface presents flat tuberosities and appears as if granulated.

Sanio-purulent pericarditis occurs very rarely, now and then in connection with ichorous and cancerous processes which have extended from adjoining structures, for instance, with cancer of the œsophagus, sloughing inflammation of the mediastinal lymphatic glands, or when foreign bodies (pieces of bone) penetrate from the œsophagus into the pericardium.

TUBERCULOSIS OF THE PERICARDIUM.

Tuberculosis of the pericardium occurs either as acute disseminated miliary tuberculosis or as tuberculous inflammation; in the former variety the miliary tubercles are found deposited chiefly at the base and along the vessels.

Tuberculous pericarditis frequently causes at the same time an eruption of tubercles and a fibrinous hemorrhagic exudate; after detaching the delicate or massive, reddish colored, fibrinous deposits, the miliary tubercles are found in the injected serosa.

Tuberculous pericarditis occurs chiefly in old persons and in children; in the latter especially in connection with tuberculosis of the intrathoracic glands.

HYDROPERICARDIUM.

(Dropsy of the Heart.)

There is an accumulation of a watery, clear, wine-colored fluid in the pericardial sac, which is correspondingly dilated. The slighter grades (15–30 c.c. =fl. ℥ ss.-i. of fluid) are quite frequent; more pronounced cases (80–100–200 c.c. =fl. ℥ iiss.-iii.-vi.) are found in at least ten to fifteen per cent of all autopsies. The serosa in this condition is smooth, glossy, and transparent.

Hydropericardium develops in numerous diseases of the heart and lungs, in hydræmic conditions—tuberculosis, carcinoma, chronic nephritis, or as a concomitant state in general dropsy.

HÆMOPERICARDIUM.

A pericardial effusion of blood in variable amount is found most frequently with gunshot and stab wounds of the pericardium and heart, with spontaneous rupture of the heart, and with perforation of an aneurism of the aorta into the pericardium.

DISEASES OF THE HEART MUSCLE.

HYPERTROPHY AND DILATATION.

The normal heart of an adult man is said to correspond in its circumference to about the size of his fist.

The heart is considered to be hypertrophied when its bulk exceeds the maximum which may be attained by the most powerful physiological work (hard labor, vigorous muscles of the body). The relative weight of the heart (proportion of the weight of the heart to that of the body) in a healthy person is as 1 : 200; with a medium weight of the body of 60 kgm. =132 lb., that of the heart is about 300 gm. =10½ oz.

The hypertrophy is either diffuse, involving the entire heart, or it affects only certain portions, for instance, the right ventricle and auricle alone, or it is confined to the left ventricle.

The causes are usually mechanical, for instance, valvular defects or disturbances in the lesser circula-

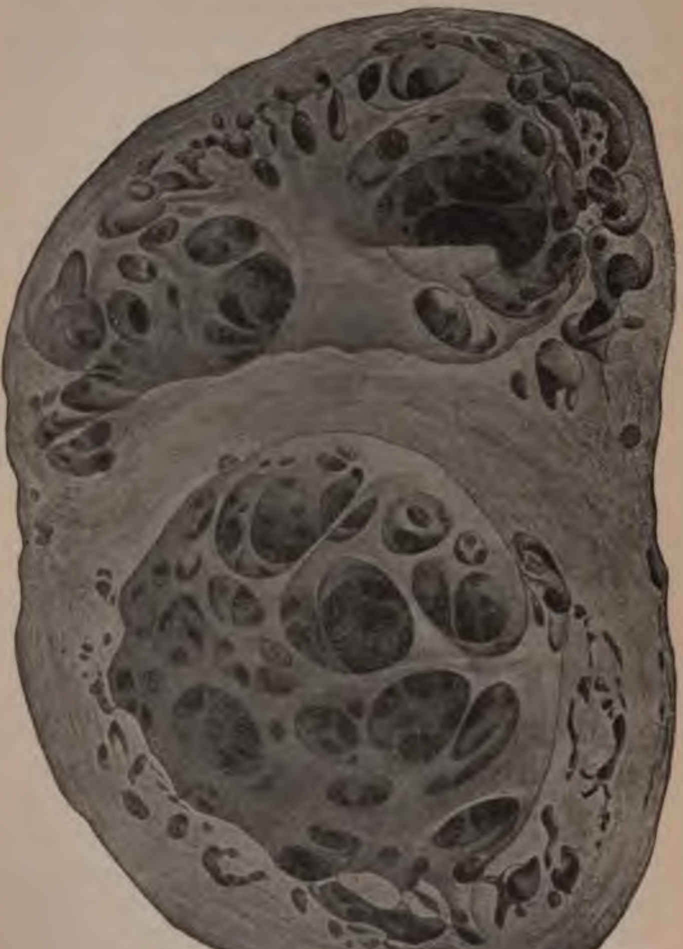


FIG. 1 *a*.

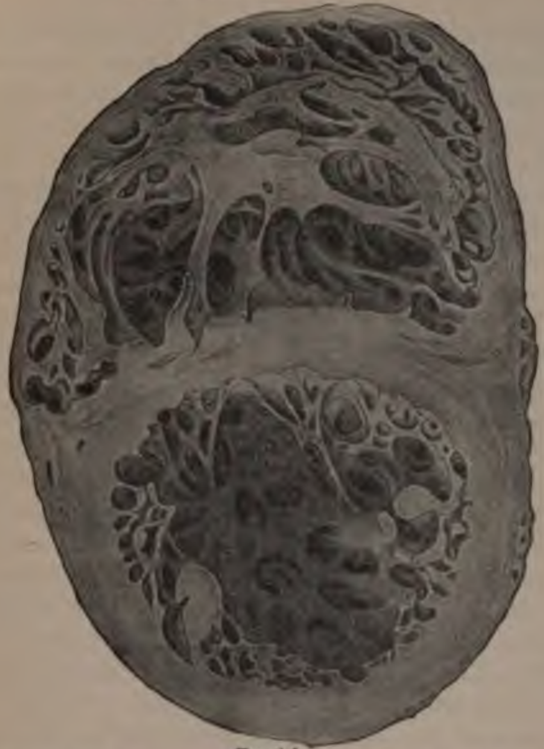


FIG. 1 *b*.

FIG. 1 *a* and *b*.—Idiopathic Hypertrophy and Dilatation of the Heart. Transverse section of the heart, injected with alcohol in the cadaver; the normal heart being placed alongside for comparison.

Anatomically this disease is characterized by the nearly uniform implication of both ventricles. The muscle is firm, rigid, usually of a dark-red color; the trabeculæ and the papillary musces are powerfully developed throughout. The primitive muscular bundles appear widened, perhaps also numerically increased, and the connective-tissue stroma is likewise increased. The valves are normal.

The weight of the heart rises from 300-350 gm. = 10½-12⅓ oz. (normal) to 540-600 gm. = 19-21 oz. and more. Idiopathic hypertrophy and dilatation affect usually powerfully built and well-nourished persons, who are often distinguished by great width of the arteries and an abundance of blood. When a fatal issue results, which is due to heart failure, the average age is forty to forty-three years. The large majority of the cases belonging to this category may be explained by the habitual excess in the consumption of beer or wine in connection with true plethora; the toxic effect of the alcohol, the physical action of the large amounts of fluid, and finally the nutritive qualities of the beer being factors in the result. Associated excessive muscular labor and bodily exertions often favor the development of this cardiac hypertrophy.

tion (reduced size of the lung); hypertrophy of the left ventricle is found mainly with chronic nephritis, sclerosis of the aorta and of the arteries in general.

The so-called idiopathic hypertrophy of the heart, Fig. 1 *a* (usually combined with dilatation), is most common in certain forms of chronic alcoholism (beer drinkers), in which plethora (high living) and toxic influences are active pathogenic factors. This diagnosis is admissible only when the ordinary anatomical causes of cardiac hypertrophy (valvular disease, arteriosclerosis, nephritis, pulmonary disease, etc.) are absent.

Hypertrophic portions of the heart are generally of firm consistence, sometimes having a board-like feel, and the trabeculæ are strongly developed. The thickness of the walls being markedly influenced by the size of the cavities, the most reliable mode of determining the hypertrophy, *i.e.*, the increased volume, is that of weighing the heart after suitable cleansing of the chambers.

Very often the hypertrophy of the muscle is combined with dilatation (excentric hypertrophy) of the chambers of the heart; both anomalies are in many ways co-ordinated, being due to the same factors—plethora, increased quantity of blood, and toxic effect upon the heart.

Simple dilatation results from increased internal pressure and is followed by a corresponding thinning of the wall; or else the dilatation depends upon a diminished tone and reduced resistance of the wall (passive form); it is met with in atrophy of the muscular structure, in degenerative conditions (fatty degeneration), prolonged anæmia, and high fever.

Aside from the diffuse hypertrophy which affects all the parts of the heart uniformly, the right or left half of the heart is frequently found alone involved.

Dilatation and hypertrophy of the right ventricle and auricle are often met with in restriction of the pulmonary circulation, in emphysema of the lungs, in diminution and shortening of the thoracic space by scoliosis and kyphosis, and in stenosis and insufficiency of the mitral valve; while hypertrophy and dilatation of the left ventricle occur especially in chronic nephritis (renal hypertrophy of the heart), and in sclerosis of the arteries (vascular hypertrophy of the heart).

The fatal issue in the various forms of cardiac hypertrophy, particularly also in the idiopathic variety combined with dilatation, after a progressive existence extending over years, is due in the majority of cases to weakness and exhaustion of the overworked muscle, which need not show any terminal degeneration under the microscope.

MYOCARDITIS.

Acute parenchymatous myocarditis occurs particularly with acute intoxications and acute infectious diseases having a marked febrile character. The muscle in such conditions appears relaxed, discolored to a grayish-red, and its consistence is diminished. Under the microscope we find in place of the normal transverse and longitudinal striation a finely granular opacity, which at many points merges into the first stages of fatty degeneration.

The recovery from the causal disease goes hand-in-hand with the retrocession of this process and the healing of the affection of the heart muscle; or else higher degrees of fatty degeneration occur, which greatly impair the function of the heart and may end fatally (fatty heart, fatty degeneration of the heart muscle).

Interstitial myocarditis is met with as an acute infectious and purulent inflammation, either by extension of infectious mycotic processes from the valves (endo-myocarditis) or by metastasis and embolism in septico-pyæmia.

Chronic interstitial or indurated myocarditis (Fig. 29) is mostly localized, though sometimes it is spread over large portions of the heart; when asssociated with or following destruction and disintegration it causes atrophy of the muscular elements of the wall and the formation of fibrous tissue—callous induration. It often results from circumscribed disturbances of circulation, in connection with sclerosis and contraction of the coronary arteries, and with embolism and thrombosis of these vessels associated with anæmic necrosis and hemorrhages. In place of the muscular bundles which have been deprived of nutrition or insufficiently supplied with blood a callous connective tissue develops by way of regeneration. Particularly at the tip of the left ventricle we find, as the terminal condition of such local circulatory disturbance and of the necrotic malacia caused thereby, extensive indurated masses with partial bulging of the lumen of the ventricle (chronic partial aneurism of the heart), whose interior is frequently covered with older, laminated thrombi situated at the wall. Stenosis and occlusion of the coronary arteries, by reason of the disturbed nutrition, causes either the formation of cardiac indurations or, when larger branches are affected, anæmic necrosis and rupture of the heart.

Fibrous indurated myocarditis is sometimes due

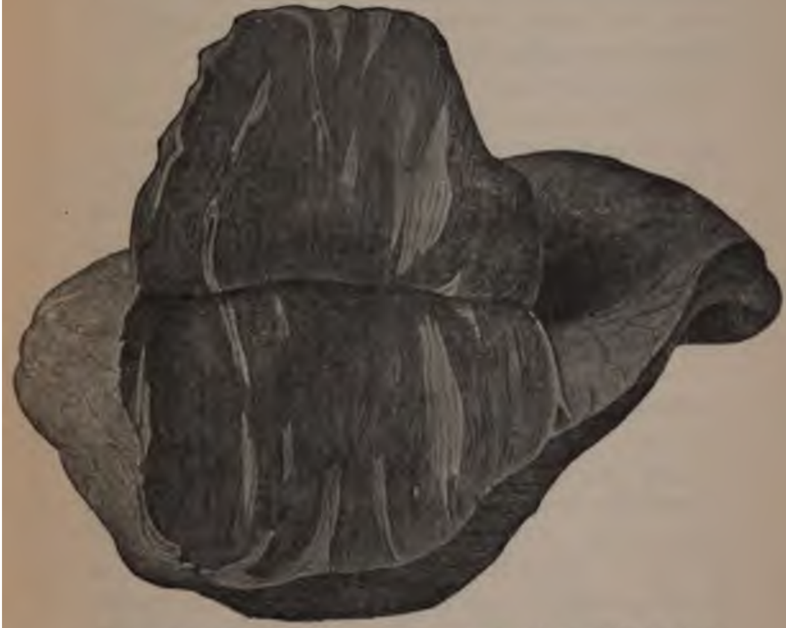


FIG. 2.—Indurated Myocarditis. Chronic interstitial myocarditis. Scattered through the heart muscle are streaky, whitish discolored masses of firm consistence, composed of fibrous connective tissue. These indurations are the product of a chronic interstitial inflammation whose cause in many cases cannot be determined. In a more circumscribed form we find callous metamorphoses of the muscular wall, particularly at the tip of the left ventricle and at the ventricular septum in sclerosis of the coronary arteries; in which case the masses of connective tissue represent the process of recovery from an embolic or thrombotic myomalacia, from an anæmic and hemorrhagic necrosis with secondary formation of indurations and cicatrices.

to syphilis or develops in connection with acute rheumatic polyarthritis—endo-myo-pericarditis fibrosa.

Purulent or infectious myocarditis is comparatively rare; in embolic septico-pyæmia the heart muscle is sometimes interspersed with miliary abscesses. Now and then an ulcerative necrosing myocarditis starts from the heart valves when a similar malignant process has become localized upon them.

FATTY HEART.

This is met with in different forms, *i.e.*, as fatty degeneration of the muscle or as an excessive deposit of fat in the subepicardial connective tissue with proliferation of fat cells (fat-containing connective-tissue cells) between the atrophying muscular elements—fatty infiltration of the muscle.

Fatty Degeneration of the Heart.

Fatty degeneration of the heart muscle develops sometimes very rapidly in acute poisoning (with phosphorus), when it may attain great intensity in a few days. More frequently it arises gradually; at first it usually occurs in patches which by degrees spread diffusely. The subendocardial and subepicardial segments of the heart muscle are as a rule most seriously involved. It is useful to distinguish three grades:

(1) Slight fatty degeneration. While the transverse striation is preserved we observe a deposition of minute fat droplets and granules in moderate quantities. This variety is found in many acute and

chronic diseases; altogether in nearly one-fifth of all cadavers.

(2) Moderate fatty degeneration. The transverse and longitudinal striation is but faintly indicated; the several droplets of fat are larger and separated from each other. This variety can be demonstrated in grave acute infectious diseases.

(3) Extreme fatty degeneration (Plate 2 *a*). The transverse and longitudinal striation has disappeared; the muscular bundles appear like tubes filled with fat; the drops of fat are very numerous and large, reaching the size of a red or white blood corpuscle. In the latter case the internal surface of the chambers of the heart appears spotted like a tiger skin. This variety occurs especially in chronic diseases of the heart and in tuberculosis. The contained fat rises from about eight or ten per cent of the solid constituents to twenty-two to twenty-five per cent.

The heart muscle is at the same time friable, sometimes of the consistence of butter, colored dull gray and pale yellowish; the cavities are dilated and the wall is frequently correspondingly stretched and thinned.

Cor Adiposum.

Adipositas s. Obesitas Cordis (Plate 2 *b*).

The true fatty heart is characterized by excessive development of the subepicardial adipose tissue, which in the higher grades envelops the muscular structure like a capsule, so that the red muscular wall is not at all visible externally.

In stout, well-nourished persons there is present beneath the epicardium, especially at the base of the heart, a certain quantity of fat which, when the muscular structure is properly developed, is within the normal limits. In general obesity, particularly the form which develops under the favoring influence of alcohol, we often find pathological varieties of fatty heart, characterized by an enormous increase of the fatty envelope of the heart, whose thickness may reach 0.5–1 cm. = $\frac{1}{5}$–$\frac{2}{5}$ inch. The demarcation of the fatty layer from the muscle, which is normally quite sharp, is obliterated; the fat proliferates between the muscular bundles, displacing them, and secondarily produces atrophy of the muscular substance, which is especially marked at the right ventricle.

Aside from general obesity and alcoholism, another variety of fatty heart occurs in anæmic, chlorotic, and cachectic subjects (particularly in cancer patients), *i.e.*, the cachectic fatty heart. In this variety we observe primary atrophy of the muscular structure and secondary proliferation of fat, as it were *ex vacuo*.

ATROPHY OF THE HEART.

Atrophy of the heart should be clearly differentiated from hypoplasia of the organ; the latter consists in defective development and abnormal smallness of the heart, present since childhood. It is usually associated with abnormal narrowness and thinness of the aorta and the larger vessels, also with chronic anæmia and chlorosis—a condition which is generally connected with stunted bodily development.

True atrophy of the heart, which rarely reaches a high degree, is characterized by diminution of volume and weight (to two-thirds to one-half of the normal), tortuousness of the vessels passing under the epicardium, abnormal narrowness of the chambers (concentric atrophy). In the majority of cases, particularly in men of middle and advanced age, the firm muscle is at the same time dull brownish discolored; this is due to the deposition of numerous golden yellow masses of pigment in the region of the muscle nuclei (brown atrophy, Plate 3). In senile atrophy there is always some hyperplasia of the connective tissue, which is especially marked in the auricles (senile fibromatosis). In the atrophy observed in connection with fatty degeneration (fatty atrophy) the muscle is at the same time of a dull yellowish-gray color, often spotted like a tiger skin, usually with dilatation of the chambers (excentric atrophy). The atrophy of the heart muscle in adipositas cordis, in which connective tissue containing fat cells develops between and in place of the atrophying muscular bundles, so that the muscle appears streaked with fat like bacon, has been discussed above.

Mention might here be made of sclerosis of the coronary arteries occurring especially in men above forty-five years of age, either as a local phenomenon of general arterio-sclerosis or confined to the coronary arteries. These vessels feel like hard cords; when dissected out they appear as stiff, quill-like tubes with contracted lumen; their walls are usually irregularly thickened and contain deposits of lime. The heart muscle may present different conditions; now and then sclerosis of the coronary arteries seems to develop in a hypertrophied heart; more frequently we find secondary atrophic states of the ventricular walls, indurations, chronic aneurisms of the heart, and finally diffuse dilatation of the ventricles. Clinically the picture presented is that of angina pectoris or stenocardia (attacks of cardiac dyspnœa, arrhythmia, and violent pain).

Endocarditis.

(Inflammation of the Endocardium.)

This affection is localized by preference on the valves (valvular endocarditis), more rarely on the parietal investment of the chambers of the heart (parietal endocarditis).

There are numerous forms: acute, subacute, chronic, and recurrent endocarditis. From an etiological point of view we distinguish idiopathic primary endocarditis from the secondary form, which develops either as infectious and metastatic or by continuity from the surrounding structures (for instance, extending from the intima of the aorta to the aortic valves). Finally we differentiate benign, noninfectious forms from the infectious, the chief representative of which is the septico-mycotic, ulcerative endocarditis. According to the products we distinguish productive and deforming inflammations, *i.e.*, endocarditis verrucosa, fibrosa, calculosa, and retrahens; also the destructive septic form, malignant or diphtheroid endocarditis, with various mixed and transitional forms. In the following remarks we confine ourselves to the description of the most important forms.

Infectious Endocarditis (Plate 4).

(Endocarditis Maligna, Ulcerosa.)

This occurs usually acutely, either primarily and cryptogenetically or secondarily and metastatically in septic wound infection, puerperal sepsis, etc.

The cause, as a rule, is the deposition and multiplication of septic germs (staphylococci and streptococci). The sites of predilection are the valves of the left ventricle, also chronically diseased valves for the form of recurrent endocarditis; the primary localization is especially apt to be the line of contact and closure of the valves, so that we are justified in assuming that mechanical lesions of the endothelium produce a sort of local predisposition which greatly favors the deposition and multiplication of the pathogenic germs. The frequency may be stated as about 0.6 per cent of all cadavers; seven to eight secondary cases to one primary; twenty-five per cent of all cases belong to the class of recurrent endocarditis, that is to say, the infectious germs develop with special preference upon diseased valves. Four-fifths of all cases of septic diphtheroid endocarditis occur in the left heart, especially at the aortic valves; only one-fifth affect the valves of the right heart.

In the beginning we observe slight roughness and unevenness of the first-affected portions of the valves, minute defects and lesions of the endocardium, whose bottom and surroundings are characterized by a discolored, dull gray appearance. The delicate valve tissue becomes opaque, rotten, and friable; some constituents of the blood are rapidly deposited in the form of parietal thrombi (thrombo-endocarditis). Owing to the destructive and ulcerative tendency, the affected portions break down or the valves are abnormally distended and bulging, with the formation of an acute aneurism and a tendency of the valves to break down and to become perforated. The thrombotic deposits likewise, by reason of their mycotic infiltration, present a discolored, dull gray, and greenish-gray appearance; they are very brittle and, like the rest of the destroyed valves, easily give rise to emboli.

The infectious and ulcerative process has, moreover, a tendency to extend to the adjoining regions; secondary purulent and ulcerative myocarditis and pericarditis are often observed, and are particularly apt to spring from the aortic valves. Besides the local functional disturbance in the form of insufficiency and stenosis of the valves, we observe multiple embolic processes in the peripheral organs of the body, especially in the spleen and the kidneys; in short, the clinical and anatomical picture of embolic septicopyæmia.

The process generally ends fatally, more rarely there is a tendency to a subacute and chronic course; in the latter case intermissions and exacerbations occur almost regularly.

Rheumatic endocarditis, met with as a local manifestation of acute articular rheumatism, is evidently also infectious in its origin, and should perhaps be described as a septiform inflammation. In view of the largely benign course of acute polyarthritis, such cases of valvular disease are extremely rare on the post-mortem table; more frequently we observe, after many months or years, the chronic results and residues of the acute infection. In some cases of asserted fatal articular rheumatism a mycotic-bacillary septic valvular disease has been found; in such instances the trouble was evidently some septic disease, some general infection presenting the appearance of an acute febrile articular rheumatism (rheumatoid septic endocarditis and polyarthritis). The polymorphous course of the process is to be explained, therefore—aside from the influence of the individual predisposition—by the variability and the degree of virulence of the germs of infection and by the transitional forms to septic infection.

The infectious rheumatic form most probably runs its course as a verrucose endocarditis with the formation of secondary thrombi—at the line of closure of the valve we observe delicate gray and grayish-red tubercles which appear moniliform along the line of closure after opening the valve; they gradually increase in size until they finally form larger coxcomb-like proliferations, consisting partly of newly formed young connective tissue, partly of thrombotic deposits. A slow course and a tendency to relapses are often observed. A sharp distinction between verrucose and ulcerative endocarditis is not feasible either on anatomical or on bacteriological grounds. Either form, when terminating in incomplete recovery, may change into chronic fibrous endocarditis.

presents frequently on the thickened valve tissue whitish and grayish-red granules, verrucæ, varying in diameter and sometimes covered with thrombi. In the older, long-standing cases it is difficult without the aid of the microscope to distinguish between the thrombotic, partly calcified deposits and the inflammatory exudate. The ostium of the valve, which is generally markedly stenosed, appears sometimes surrounded by mortar-like masses in which the valve cups have practically perished (endocarditis calculosa).

The leaflets of the auriculo-ventricular valves, particularly those of the mitral, are gradually transformed into indurated firm masses which coalesce and at last have an almost cartilaginous feel; the tendinous cords become progressively shorter and thicker and attain the diameter of a knitting-needle and above (endocarditis retrahens).

In the semilunar valves, especially those of the aorta, we find in this disease, along with thickening of the free margins, which look as if rolled over, or of the entire valves, a corresponding shrinkage and shortening of the valve tissue, frequently also a coalescence of the several leaflets.

As the indurative process extends also to the ring of attachment of the valves it has a constricting effect upon the lumen of the ostium, and thus arise different degrees of stenosis of the latter. While the normal ostia of the heart of an adult man admit at least the index finger or the thumb, in the endocarditis here described we observe the ostium changed into a slit-like opening with rigid margins, which in advanced cases hardly admits a medium-sized lead pencil.

Owing to the rigidity and shortening of the valves, various degrees of insufficiency (valvular insufficiency), with or without stenosis, also develop.

At the aortic valve additional factors favor the insufficiency caused by the valvular anomaly. Owing to the frequently coexistent and primary aortic sclerosis (Plate 8) the ascending aorta is distended, causing dilatation of the aortic ostium. The insufficiency resulting primarily from the moderate thickening and shortening of the semilunar valves is associated with a relative insufficiency, and thus we see before us a combined or potential valvular insufficiency, which is sometimes increased still more by the coexisting dilatation of the left ventricle.

Besides this accessory relative insufficiency there is a relative insufficiency which occurs rather frequently in consequence of dilatation of the right ventricle and auricle at the tricuspid valve. With the widening of the cavities and of the ring of attachment of the valve there are increasing tension and stretching of the leaflets, tendinous cords, and papillary muscles of the tricuspid valve until the latter at last is no longer able to close the ostium, which is sometimes dilated to double and treble the normal.

A third form of insufficiency of the auriculo-ventricular valves is known as muscular; it is due to fatty degeneration or indurative metamorphosis of the papillary muscles and is met with secondarily in fatty degeneration of the heart muscle, which shows a special predilection for the papillary muscles and may attain marked degrees. It is found also in anæmic and chlorotic patients in whom the nutrition of the heart muscle suffers. To this class belong the cases in which murmurs are audible during life and are ascribed to a lesion of the valves, which, however, are seen to be perfectly normal at the autopsy.

That valves affected with chronic disease form a locus minoris resistentiæ, that relapses occur (endocarditis recurrens, Plate 5, Fig. *a*), and that occasionally also infectious septic processes are localized by preference upon diseased valves, has been stated above.

Finally, it is readily understood that the inflamed heart valves often give rise to emboli which penetrate from the valves of the right heart into the lungs, and from those of the left heart into the major circulation (brain, kidneys, spleen [Plate 15], and the arteries of the lower extremities). In benign endocarditis such emboli produce merely a mechanical effect, causing various disturbances of the circulation, infarctions, and neurotic processes; in malignant infectious endocarditis they give rise to metastatic abscesses, in short to the picture of embolic septico-pyæmia.

THROMBOSIS OF THE HEART.

(Plate 5, Fig. *b*.)

The chambers of the heart are more frequently the seat of thrombi than is any other part of the vascular system.

Aside from the secondary thrombotic thickenings and deposits in valvular and parietal endocarditis, we find in numerous, especially chronic diseases marasmic thrombi as terminal and preagonal products; these are particularly important in estimating the vital energy of the heart, but they are so more for the pathological anatomist than for the clinician. They are whitish or grayish-white, at least at first, and are situated at the wall, especially in the indentations of the right ventricle, and intimately interwoven with the trabeculæ; their size ranges from that of a pin's head to that of a hempseed or pea, they are roundish in form, and either solid or contain in their interior a whitish-gray pyoid mass (puriform deliquescence). In the auricles they frequently attain a larger size, up to that of a cherry or plum; now and then they are laminated and mixed with the constituents of the blood, *i.e.*, mixed thrombi. In the left ventricle and auricle they are less frequent, and when present are located chiefly at the wall of the apex.

In the development of marasmic thrombosis in the cavities of the heart the mechanical factor, the defective circulation, evidently plays the chief rôle; under the influence of heart failure or cardiac marasmus stasis occurs in the indentations of the chambers; whether a lesion of the endocardium, defects of the endothelium, is a concomitant factor in their development is still undecided. On the other hand, the formation of marasmic thrombosis is certainly favored by alterations in the composition of the blood (leucocytosis, infectious processes, etc.).

DISEASES OF THE ARTERIES.

Arteriosclerosis, Chronic Endarteritis. Atheroma.

(Plates 8, 9, 10, and 11.)

The intima of the arteries contains no vessels—a peculiarity which largely influences the course of inflammatory processes; swelling and functional disturbance are the main characteristics of such processes.

In the aorta chronic endarteritis begins with the development in the intima of flat, garden-bed-like thickenings which differ from the surrounding parts by their more opaque and yellowish color. They consist of a gelatinous connective tissue, associated with which are accumulations of cells that exhibit a special tendency to fatty degeneration. In this way small shallow depressions are formed, which contain, when the process is farther advanced, minute granules of fat, cholesterin crystals, and particles of lime. As such atheromatous foci gradually disintegrate the remaining layers of the intima, erosion, perforation toward the lumen of the vessel, and the formation of an atheromatous ulcer result. When this process occurs simultaneously at different points of the intima, the latter acquires a cribriform perforated appearance. At such ulcerating points small parietal thrombi form, which give rise to embolic occlusions. In older subjects, moreover, we often find a tendency to calcification of the hyperplastic portions, largely also a simple calcareous degeneration of parts of the wall which were previously normal. In that event we observe embedded in the wall true calcareous plates which coalesce at many points; in place of the elastic and contractile vessel wall there is a rigid tube which creaks when divided with scissors. The chief characteristics of this grave process, therefore, are swelling, cellular and connective-tissue thickening having a marked degenerative tendency, in the shape of fatty metamorphosis and calcification. All the stages of the process are as a rule found side by side—from the spotted yellowish opacity and garden-bed-like thickening to calcification and ulceration. The intima in consequence appears truly deformed—endarteritis deformans. Owing to the loss of elasticity and contractility with atrophy of the muscular coat, diffuse and circumscribed dilatations of the artery develop, *i.e.*, ectasiæ and aneurisms. In the medium-sized and smaller arteries the parietal disease causes stenosis of the lumen.

A special form of chronic endarteritis is that occurring at an earlier age (from thirty to forty or forty-five years), the gummous form due to syphilis. The intima of the aorta contains flat, garden-bed-like, spotted elevations of a grayish-white color which have no marked tendency to fatty degeneration and calcification. Postsyphilitic arteritis, which extends gradually from the adventitia to the intima and presents no tendency to fatty degeneration and calcification of the diseased parts, is also observed in a very characteristic form as a cellular infiltration of the cerebral arteries in persons of a comparatively early age (between twenty-five and forty-five years) who had acquired syphilis some longer or shorter time previously and had apparently recovered. This form often terminates fatally by obliteration of the lumen of the vessel (endarteritis obliterans), followed by softening of the brain or apoplexy.

The causes of arteriosclerosis which occurs in part as a senile disease are obviously manifold; aside from high living, alcoholism, syphilis, physical and mental overstrain, its development seems to be favored in some cases by a certain hereditary predisposition. The same injurious influences which give rise to these chronic hyperplastic and degenerative processes of the arterial wall are also largely active in producing disease of the heart. Functional overexertion (Rokitansky) is also likely to cause, in predisposed subjects, arteriosclerosis in some regions (the arteries of the brain, of the lower extremities, and the ascending aorta). In persons who exhaust their powers prematurely by overexertion and perhaps expose themselves also to other detrimental influences (alcoholism, high living) arteriosclerosis is more frequent than under opposite conditions.

The thoracic aorta, especially its ascending portion, is among the most frequently affected by the inflammatory and sclerosing process. From the ascending aorta, the inflammation extends very often to the adjoining aortic valves. The thickening and shortening of the valves, with the frequently coexisting dilatation of the ostium of the aorta, cause minor or major degrees of aortic insufficiency, which is met with especially in men of middle and advanced age, develops slowly, and no acute articular rheumatism can be traced to explain the valvular disease. In such cases of apparently primary valvular affection the starting-point of the disease is the chronic endarteritis of the ascending aorta, whose progressive character is clearly marked.*

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* Among 1,800 dissections of adult cadavers sclerosis of the aorta associated with disease of the aortic valves was found in 136 subjects, in 25 of these with marked insufficiency and in 2 with stenosis of the ostium of the aorta. In slight aortic sclerosis or when the process is located in the descending and the abdominal aorta (48 cases) the heart valves are unchanged. The sclerosis of the ascending aorta with secondary insufficiency of the aortic valves here discussed is found in men more than twice as often as in women; the age of the male patients was between thirty-three and fifty-one years; the average age of the men dead

The sequelæ of arteriosclerosis are very manifold.

Owing to the loss of elasticity and contractility, the arterial pressure rises, with secondary dilatation and hypertrophy of the left ventricle; the diseased vessels show a tendency to a diffuse and circumscribed dilatation of the lumen, to the formation of aneurisms; the degenerative processes (fatty degeneration) are apt to cause rupture, especially apoplexies in the brain. In the medium-sized and smaller arteries the lumen becomes contracted and thrombi form, with nutritive disturbances of the peripheral organs —arteriosclerotic atrophy of the brain and of the kidneys.

A special importance attaches to sclerosis with fatty degeneration of the cephalic vessels, which gives rise to presenile atrophy of the brain, to necrotic and localized softening of the brain substance; in sclerosis of the renal arteries we find maculated contraction of the kidneys; in sclerosis of the arteries of the lower extremities senile gangrene is apt to occur; sclerosis of the coronary arteries causes atrophic and inflammatory alterations of the heart muscle with serious functional disturbances (angina pectoris s. stenocardia). Furthermore, in ulcerative and calcareous inflammation of the intima of the larger arteries we observe a special tendency to the formation of parietal thrombi, which in turn cause embolism of the peripheral arteries.

The connection between arteriosclerosis and cardiac hypertrophy may vary as follows: (1) The arterio-

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of this disease was forty-seven years, that of the women fifty-four years.

sclerosis may develop primarily and, as stated above, induce a secondary dilatation and hypertrophy of the left ventricle; this type includes especially the senile forms of arteriosclerosis which occur after the fiftieth year of life and the postsyphilitic fibrous and gummous aortitis in younger persons. Excessive physical exertions also favor its occurrence, although in animals (horses, draught oxen, and dogs) which perform hard and prolonged muscular labor this vascular disease, that occupies so prominent a place in human pathology, is almost unknown. (2) In a second group hypertrophy of the heart and arteriosclerosis develop in a co-ordinated way on the same basis; the same causes (alcoholism, high living, plethora, overexertion) produce simultaneously cardiac hypertrophy with dilatation and chronic endarteritis. (3) The third group includes those cases in which the primary hypertrophy of the heart, owing to the increased chronic pressure, causes a reaction on the part of the intima of the aorta which takes the form of a fibrous and indurated endarteritis. Especially when associated with general nutritive disturbances (chronic nephritis and albuminuria) cardiac hypertrophy exerts a marked injurious influence upon the more minute arborizations of the arteries. This is the explanation of the marked frequency of cerebral apoplexy in hypertrophy of the left ventricle, whose pathogenic influence (chronic increase of the arterial pressure) is in many cases of apoplexy of greater importance than the degeneration of the cerebral arteries, which is sometimes slight. In the distribution of some arteries (for instance, in the kidneys) the hypertrophy of the heart gives rise to compensatory sclerosis and thickening of the walls of the smaller arteries, in consequence of the chronic mechanical overstrain.

ANEURISM OF THE ARTERIES.

(Plates 8, 10 and 11, 12.)

By aneurism we understand a dilatation of the arterial tube that either extends over larger portions of the vessel or is confined to a sacculation at a certan point; in the latter case the communication between the lumen of the artery and the sacculated diverticulum forms a kind of ostium which has usually a smaller diameter than the aneurismal sac proper. The diffuse, sometimes spindle-shaped or cylindrical dilatations (fusiform aneurisms) affect all the parts of the vessel wall almost uniformly, while the sacculated aneurisms (Plates 8 and 12) arise usually from a circumscribed sudden lesion of the media, *i.e.*, aneurisms by rupture.

Aneurisms vary greatly in size: on the smallest arterial twigs they rarely exceed a millet seed (miliary aneurisms), on the thoracic aorta they often attain the size of a fist, of a child's head, and more.

Aneurism develops most frequently upon the thoracic aorta (six-sevenths of all internal aneurisms), the sites of predilection being the ascending portion and the arch. The slighter grades and initial stages of dilatation of the aorta are frequently discovered incidentally in persons above the age of fifty and sixty years. The wall of an aortic aneurism presents as a rule more or less advanced grades of chronic deforming inflammation. Aside from irregular thickenings, especially of the intima, we observe, particularly at the point of greatest dilatation, extreme thinning of the wall, irregular defects of the media and intima, so that the wall consists largely only of the slightly thickened adventitia, whose resistance to the blood pressure seems to be reinforced by deposits of laminated parietal thrombi.

The fundamental condition of this important change in form of the artery is evidently arteriosclerosis which causes weakening and partial atrophy of the media. Next to senile and presenile endarteritis it is probably postsyphilitic endarteritis (endarteritis sclero-gummosa), and in a few cases also traumatic influences, that cause a development of aneurisms. Sudden increase of the blood pressure and great overburdening of the vessel wall, resulting from excessive muscular labor, may act in some cases as an exciting cause. That mechanical influences favor the development of an aneurism is proved by the frequency of its occurrence on the popliteal artery, in which a local arteritis due to the continually alternating extension and flexion produces the dilatation. In favor of the view, which has been disputed by many, that chronic inflammation of the intima with secondary mesarteritis may cause the development of an aneurismal dilatation independent of other factors, we may cite an experience from comparative pathology. In horses in which aneurisms of the aorta and other arteries resulting from sclerosis of the vessel wall practically never occur, we find on the main trunks of the mesenteric arteries (arteria mesenterica anterior) very frequently—in more than ninety per cent of all horses —aneurisms whose origin must unquestionably be ascribed to a zoö-parasitical and verminous endarteritis and mesarteritis. The importance of sudden or oft repeated increase of blood pressure in the causation of aneurisms is shown by the fact among others that these morbid changes occur twice as frequently in men as in women, though other factors (alcoholism and syphilis) may have a share in their production.

In general, aneurism resembles a slowly growing tumor which displaces, compresses, and more or less erodes neighboring organs; neither soft parts nor bones are able to resist it long. Every aneurism possesses the potentiality of progressive growth; the course is slow and the beginning of the dilatation can rarely be determined; the average duration is said to be between one and four years. In the younger patients with a vigorous heart the growth is usually more rapid. Death ensues from intercurrent diseases, sometimes from impaired pulmonary circulation or disturbed respiration. A frequent and uniformly fatal issue is by rupture, which occurs during some temporary increase in cardiac activity with resulting rise of arterial pressure, when internal hemorrhage is the direct cause of death.

Aneurisms of the thoracic aorta perforate by preference into the pleura, pericardium, trachea, lung, and œsophagus.

In the peripheral arteries aneurisms are quite rare, the popliteal artery forming an exception. In the distribution of the cerebral arteries we often find very small aneurismal dilatations—miliary aneurisms—which are regarded by many as the starting-points of spontaneous hemorrhages of the brain, occasionally also larger aneurisms from the size of a pea up to that of a hazelnut, due to arteriosclerosis, syphilitic arteritis, or occurring without demonstrable cause (possibly traumata?).

Purely degenerative processes are found in the arterial walls as follows:

1. *Fatty degeneration*, especially in the smaller arborizations of the cerebral arteries and also in the connective-tissue cells of the intima.

2. *Calcareous degeneration of the media*, in the medium-sized and small arteries of the extremities; its characteristic feature is the circular calcareous deposit which corresponds to the course of the circular muscles; this form is often included with inflammatory sclerosis and mistaken for it.

3. *Amyloid degeneration*, occurring chiefly in the minute arteries of the abdominal organs—the kidneys, spleen, liver, and intestine—and associated with amyloid degeneration of the parenchyma of the organs named.

INJURIES OF THE ARTERIES.

Small wounds of the arteries heal, without material thrombosis and without obliteration of the lumen, by union and adhesion of the wound margins, the blood circulation remaining undisturbed. After complete division of arteries (amputations), under the favoring influence of retraction and contraction of the severed vessel or the ligature, thrombosis occurs and at first occludes the lumen as far as the nearest proximal branches; soon after the thrombus is replaced, by way of substitution, by young connective tissue which springs mainly from the intima (so-called organization of the thrombus).

In lateral injuries of the arterial trunks, especially those of the extremities, the blood escapes into th surrounding tissue, which is displaced; the injure point is covered with a mass of blood like a tumo which pulsates, becomes gradually encapsulated, an is known as a false aneurism (aneurysma spurium).

Thrombosis of the Arteries.

This occurs either autochthonously in consequenc of disease of the wall (sclerosis, endarteritis) or c other lesions of the vessel wall, such as trauma an ligation, or else by emboli coming from a distanc Such embolic thrombosis is frequently traceable t the right heart or the peripheral veins derived fro the main trunks and branches of the pulmonary artery, or to the left heart and the trunks of the large arteries (aorta) in the distribution of the periphera arteries of the body.

As the result of the embolic occlusion of the vessel we frequently observe in the lung hemorrhagic infarctions of variable size; in the distribution of the peripheral arteries of the body with corresponding collateral circulation merely temporary ischæmia; benign embolus in such a case acts like a ligature o the vessel. In organs with so-called terminal arterie (spleen and kidneys) the result is coagulation necrosis and embolic infarctions; in the distribution of th basal arteries of the brain, ischæmic softening.

When the emboli are infectious, filled with septi germs—for instance, when they are derived from septic thrombo-phlebitis or mycotic endocarditis— they cause at the points where they are arrested with or without circulatory disturbance, septic puru lent processes, metastatic abscesses, forming the anatomical substratum of embolic septico-pyæmia.

DISEASES OF THE VEINS.

PHLEBECTASIA. VARICES (Fig. 3).

One of the most frequent anomalies of the veins is dilatation, phlebectasia.

As a rule we have to deal with disease of entire venous plexuses, whose arborizations appear dilated and tortuous, with nodular distentions and sacculations like diverticula of the walls. The media of the wall is alternately hypertrophied and atrophied, the intima is evenly thickened by a kind of fibrous endophlebitis. Veins thus affected frequently show a tendency to rupture and hemorrhage from slight causes.

The causes of the dilatation of the veins are manifold. Aside from hereditary tendency, we distinguish central causes, diseases of the heart (weakness of the heart), lungs, pleura, and liver; and peripheral causes, compression of large venous trunks by tumors, by the pregnant uterus, by the accumulation of fecal masses, unsuitable and constricting articles of dress, and insufficient muscular activity.

As a result of the dilatation of the veins there are retardation of the circulation, stasis, and the formation of thrombi; the mucous membranes within the domain of the affected part are subject to chronic catarrhs owing to the defective venous return flow; the external skin, especially that of the legs and feet, is liable to chronic inflammatory processes with excessive deposit of pigment and has a tendency to the formation of ulcers (varicose ulcers).

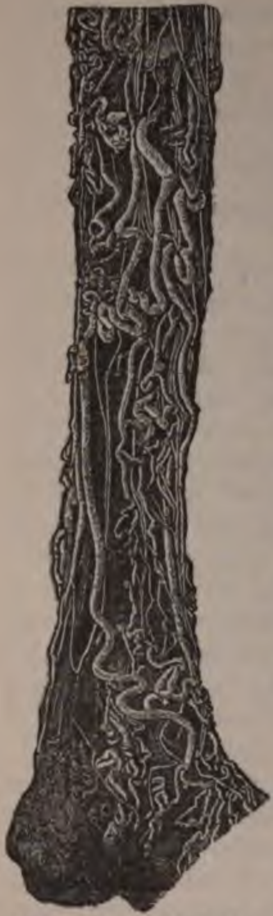

FIG. 3.—Varices of the Leg. (After v. Lesser*-Teichmann.) The subcutaneous veins of the leg are almost without exception dilated and tortuous. The dilated veins are accompanied by lymph vessels characterized by their straight course.

The regions most frequently involved are the subcutaneous venous plexuses of the leg and thigh; the pelvic veins, particularly in the neighborhood of the rectum, and the veins of the spermatic cord (varicocele). Rarely we observe phlebectasia of the subcutaneous abdominal veins, which develops in consequence of the collateral circulation in thrombosis of the inferior vena cava or of the portal vein. Quite exceptionally we may observe true varices at the base of the tongue, in the œsophagus, and in the small intestine, where these formations sometimes assume almost the character of angiomata.

PHLEBITIS. INFLAMMATION OF THE VEIN.

The most dangerous form of disease of the veins is infectious septic phlebitis, which as a rule extends from peripheral organs, such as infected wounds and puerperal endometritis, or from neighboring structures to the vessel wall, *e.g.*, from suppuration of the middle ear, with caries of the petrous portion of the temporal bone, to the dura mater of the base of the skull and the transverse sinus embedded in it. Sinus thrombosis after suppuration of the middle ear has a tendency to extend centrally to the jugular vein; in one-fourth to one-third of all cases it may terminate in recovery with or without operation; often, however, purulent leptomeningitis supervenes. Septic phlebitis resembles infectious processes of the connective tissue in general; the wall of the vein is usually infiltrated with pus, discolored, rotten, and friable; the intima becomes rough and uneven, as if corroded. Very soon thrombotic deposits are superadded, forming septic thrombo-phlebitis. In such a case the thrombosis is of secondary occurrence; on the other hand, a thrombus which has arisen in some way or has been transported in the circulation may produce a secondary disease of the wall of the vein, phlebitis. When such infectious thrombi of peripheral origin enter the circulation, metastatic embolic abscesses result.

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* L. v. Lesser: "Ueber Varicen." Virchow's Archiv f. pathol. Anatomie, Bd. 100, 1885, plate x.

A benign and productive endophlebitis is observed in the organization of thrombi, when a connective tissue rich in cells gradually replaces the coagulated constituents of the blood.

Thrombosis of the Veins.

(Plate 13.)

The formation of thrombi in the veins is frequently observed, either as secondary thrombosis in connection with parietal lesions, such as injuries and inflammation of the vessel wall, or more commonly as primary autochthonous thrombosis, which occurs especially in defective circulation, in the development of varices, and as the marasmic form.

Marasmic and autochthonous thrombosis occurs in the larger venous trunks of the lower extremities and in the sinuses of the dura mater. Any disease or process that causes heart failure either rapidly or slowly, such as acute and subacute febrile infectious diseases or those associated with cachexia and anæmia (carcinoma, chronic tuberculosis, chlorosis, the various anæmias, and the puerperium), greatly favors the development of marasmic thrombosis. The first beginning of a thrombus usually forms in the interior of the valves: the small thrombus, originally parietal, gradually becomes obstructive by enlarging in all directions. As the thrombus extends centrally from smaller into larger branches, it is a frequent occurrence for it to project like a cone into another vessel and thus to act with reference to the blood current as a parietal thrombus, from which smaller or larger particles are easily detached and carried as emboli in a central direction; in this way embolism of the pulmonary arteries occasionally results.

After a variable length of time venous thrombi undergo different metamorphoses; they may become organised by way of a productive endophlebitis, or we may observe central disintegration (puriform softening), contraction, discoloration, now and then also calcification (formation of phleboliths). Such phleboliths are sometimes discovered accidentally in the dilated venous branches of the uterus, spleen, peritoneum, and the lower extremities.

DISEASES OF THE SPLEEN.

The capsule of the spleen takes part in numerous affections of the peritoneum, especially inflammatory processes—fibrinous, purulent, and adhesive perisplenitis. Adhesions of the capsule to the surrounding organs (diaphragm, omentum) are observed with particular frequency.

Besides these have been found, usually associated with pathological processes of the splenic tissue, milky patches and nodular thickenings of the capsule, which are apt to be membranous or almost cartilaginous in older subjects with chronic enlargement of the organ.

Primary idiopathic diseases of the spleen are far more rare than secondary affections, which are often present in the form of swelling and inflammatory hyperplasia (engorgement of the spleen) with many infectious diseases (sepsis, typhoid fever, malaria, tuberculosis, and syphilis) and with circulatory disturbances (acute and chronic congestion).

The weight of the normal slpeen varies between 140 and 160 gm.=5 and 5½ oz.; length, width, and thickness measure respectively 12, 7, and 3 cm.= 4⅘x2⅘x1⅕ in.

Acute inflammatory or infectious hyperplasia of the spleen (*parenchymatous splenitis*) is of hæmatogenous origin and is explained by the rôle played by this organ as a blood filter for organized and chemical noxæ. Besides, the spleen takes up the detritus of the red and the broken-down white blood corpuscles, especially in septic blood infection and in certain intoxications (with potassium chlorate) which cause a rapid disintegration of the red corpuscles with hæmoglobinæmia and hæmoglobinuria. In acute infectious splenitis we find the organ enlarged to two, four, or five times the normal; the capsule is tense, the pulp of a brownish-red color; the consistence is diminished, sometimes pultaceous, almost deliquescent, and the organ is full of blood; this is the case in typhoid fever (280-500-700 gm.=10-17½-25 oz.), pyæmia and septicæmia (250-300-700 gm.=9-10½-25 oz.), anthrax, acute and subacute tuberculosis (170-220 gm.=6-8 oz.). In fatal croupous pneumonia, acute infectious splenitis is found in more than two-thirds of all cases, especially often in the stage of gray hepatization, in which there is an active regeneration of the cellular elements of the blood (leucocytes) in connection with the enormous loss of blood (acute hemorrhagic pulmonary exudation). In typhoid fever the enlarged spleen often contains circumscribed, dirty gray, necrotic foci, separated from the surrounding tissue by a demarcating suppuration; these probably result from the local stases and the necrosing effect of the pathogenic specific typhoid bacilli deposited in the spleen.

Chronic hyperplastic splenitis is characterized by increased firmness of the parenchyma, by proliferation of the connective-tissue elements. This form is found especially in the splenic anæmia of children, in congenital syphilis,* and in malaria.

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* The spleen of newly born children has an average weight of 9-10 gm.=139-154 gr.; in congenital syphilis it rises to 15-32, even as high as 100 gm.=½, 1, 3½ oz.

Chronic splenitis is also found in a specially characteristic type in leukæmia (splenic form), in which the organ may be enlarged to ten, fifteen, or more times the normal and attain the volume and consistence of the liver; the color is a more fleshy red, the consistence firm, the tissue bloodless (Plate 14).

Numerous variations are presented by engorgement of the spleen, which is observed, especially in the subacute and chronic forms, in many circulatory disturbances (diseases of the heart, lungs, and liver). In the initial stage the spleen appears enlarged, particularly in thickness, is full of blood, dark cyanotic in color, the framework is increased, and the consistence firm (cyanotic induration, Plate 15). When the venous hyperæmia has lasted a long time, sometimes for years, *e.g.*, in cirrhosis of the liver or valvular disease of the heart, the consistence increases steadily, the blackish dark color becomes more intense, the capsule is frequently thickened, and finally atrophy occurs, usually associated with shortening of the organ (atrophic engorgement of the spleen).

Among the circulatory disturbances the sequelæ of embolic occlusion of the arteries—*embolic infarctions* so called—occupy a prominent place by reason of their frequency. Similar to the kidney, the arterial system of the spleen is arranged on the type of terminal arteries; hence in embolic occlusion of the branches of the splenic arteries there is no corresponding collateral circulation, but infarctions form which present different types. We distinguish anæmic infarctions, when by reason of the local ischæmia early coagulation necrosis and decoloration of the affected segment develop; these infarctions are of a pale yellowish color, sharply demarcated from the surrounding tissue; they are wedge-shaped, the base being subperitoneal, the point directed toward the hilus of the spleen; the occluded vessel is to be looked for in the region of the point. The benign embolic infarctions are more rarely hemorrhagic in quality and differ from the surrounding tissue by their dark brownish-red color, greater firmness, and a certain dryness. In the further course there is a gradual contraction of the infarctions, a cicatricial retraction of the affected part, and finally we find as the residues of the arterial occlusion only whitish fibrous cicatrices which extend to a varying depth into the parenchyma of the spleen and sometimes include orange-colored, cheesy, and calcareous remains of the cicatrized tissue. Such benign infarctions occur especially with subacute and chronic endocarditis of the valves of the left heart and with ulcerative and thrombotic endaortitis.

When the emboli are of infectious origin, *e.g.*, in septic mycotic endocarditis, there is besides the local ischæmia and hemorrhage an early purulent disintegration; the yellowish-white purulent foci with their red areola are visible through the capsule of the spleen. The metastatic abscesses range in size from a pin's head to a walnut and larger; the entire organ is considerably swollen; a secondary purulent perisplenitis and peritonitis may be superadded.

TUBERCULOSIS OF THE SPLEEN.

Aside from tuberculous disease of the capsule of the spleen which forms part of tuberculosis of the peritoneum, tuberculous processes are found rather frequently in this organ. In general we distinguish two forms: (1) Acute miliary tuberculosis of the spleen as a local phenomenon of general miliary tuberculosis. In this form the organ is considerably enlarged, soft, moderately filled with blood, and dark brown in color; the parenchyma is sprinkled with exceedingly numerous, barely visible nodules. The latter are gray and translucent, often yellowish, opaque in the centre, and differ especially by this last-named peculiarity from the Malpighian corpuscles, which they otherwise greatly resemble. (2) The tuberous, more subacute or chronic form containing tuberculous foci, which is most frequent in children (Plate 16, Fig. *b*). The lesions often appear isolated as solitary tubercles in the form of yellowish cheesy patches ranging in size from that of a hempseed to that of a pea or a cherry; their marginal zone is more gray and gelatinous and slightly injected; in many instances the larger patches result from the coalescence of several neighboring nodules, *i.e.*, agglomerated tubercles.

Syphilomata (gummata) of the spleen, in the form of irregular deposits of a yellowish color and cheesy consistence, are less common.

Neoplasms of the spleen are also rare; now and then carcinomatous or sarcomatous nodules of metastatic origin are found.

Among the degenerative processes which are localized by preference in the spleen, amyloid degeneration occupies the first rank. In both forms, the diffuse and that confined to the Malpighian corpuscles, the spleen is markedly enlarged; besides, in the former variety the organ is firm, light red or dark red in color, and the cut surface has a more or less lardaceous lustre, according to the degree and the stage of the disease. The focal variety is characterized by the localization of the process in the Malpighian corpuscles, which are swollen to the size of a hempseed or a pea and lie in the shape of light gray, glassy, translucent granules, resembling boiled sago, in the red splenic tissue (sago spleen). On the addition of a solution of iodine the amyloid parts assume a dark mahogany-brown color, the normal tissue becoming straw yellow; a watery solution of methyl violet stains the diseased parts a ruby red, while the normal tissue presents a dark blue color.

DISEASES OF THE LYMPHATIC GLANDS.

The lymphatic glands are, as it were, filters for the micro-parasitic germs of infection and for foreign bodies, both of which penetrate from the periphery by way of the lymph channels. Thus, for instance, the peribronchial lymphatic glands contain invariably particles of soot and carbon which have entered through the intact pulmonary tissue, and after tattooing of the forearm with cinnabar particles of the same pigment are present in the axillary glands. In the case of numerous infectious substances, *e.g.*, the bacilli of tuberculosis, typhoid fever, anthrax, pyogenic fungi, the poisons of syphilis and gonorrhoea—the parenchyma of the lymphatic glands constitutes a fruitful field for the colonization and multiplication of the germs; in like manner the lymphatic glands participate secondarily, almost without exception, in carcinomatous disease of peripheral organs.

The lymphatic glands, similar to the spleen, react very promptly to infectious pathological processes and to such as are prone to metastasis from peripheral organs, and are of the greatest importance to the physician in forming a diagnosis. Besides these frequent secondary and metastatic pathological processes, there are also idiopathic primary infectious diseases of the lymphatic glands, especially those of a tuberculous nature, in which the germs pass through apparently normal organs or such as are prone to inflammation (mucous membranes, pulmonary parenchyma), reach the lymphatic glands, and multiply in them; thus we often observe, especially in children, primary cheesy tuberculosis of the glands of the neck or of the interior of the thorax. Particularly in the case of tuberculosis, but also in a number of benign inflammatory processes, we may note a characteristic difference in the predisposition with respect to age; while in children, especially the debilitated and poorly nourished, such diseases—both primary and secondary—of the lymphatic glands are extremely frequent, persons of middle and advanced age are far less liable to them.

The accumulation of foreign bodies in the form of particles of soot and carbon (anthracosis) is found almost regularly with advancing age in the peribronchial glands at the root of the lung. At first we see the slaty-black pigment in the reticular lymph sinus, then in the cortical follicles, and finally scattered through the entire parenchyma; in men of middle and advanced age these glands in a condition of anthracosis are usually quite black or slate-colored and harder than normal. When other heavier and more dangerous kinds of dust, mineral or metallic fragments, reach the gland tissue together with carbonaceous particles, a chronic indurated inflammation results; on section the glands are firm, almost stony, and creak under the knife. Slighter grades of infiltration with gritty particles which are associated with anthracosis are frequent and can be determined exactly only by chemical analysis of the dry substance.

Inflammation of the Lymphatic Glands.

Lymphadenitis.

We distinguish acute and chronic forms of lymphadenitis, purulent and tuberculous inflammations.

In acute inflammation the glands appear more or less enlarged, injected, juicy, full of blood, and of a medullary consistence. The hyperplastic swelling is followed either by resolution or in the case of more intense processes by purulent degeneration of the gland tissue, the formation of an abscess. The latter again may heal by inspissation and absorption, or it may involve neighboring structures or the skin and break, or it may cause larger abscesses, purulent inflammation of the cellular tissue surrounding it. Inspissation and caseation may also end in calcification, and many partial or diffuse calcifications of internal lymphatic glands accidentally discovered at an autopsy should be interpreted as the residues of such purulent processes.

Chronic lymphadenitis usually develops slowly; the enlarged lymphatic glands are hard and the parenchyma shows a considerable increase of the reticulum with corresponding loss and atrophy of the lymphoid cellular elements. At last the gland consists of firm fibrous tissue, *i.e.*, it is in a condition of chronic induration; this alteration takes place particularly in the bronchial and mediastinal glands in consequence of anthracosis, chalicosis, and siderosis.

TUBERCULOSIS OF THE LYMPHATIC GLANDS.

Tuberculosis is probably the most important and at the same time an extremely frequent disease of the lymphatic glands.

Tuberculous lymphadenitis occurs as a primary affection—especially in children and young people—or secondarily with primary tuberculosis of the lungs, intestines, bones, and joints. The glands are enlarged; on the cut surface we see comparatively seldom true tubercles scattered over it; more often the gland seems to contain irregular cheesy foci, recognizable by their yellowish-white color and their dry and brittle consistence; sometimes partial purulent disintegration is likewise present. In a more advanced stage the enlarged glands are cheesy throughout, yellowish in color, and the lustreless cut surface resembles that of a raw potato. In the form of a primary tuberculosis this glandular affection occurs with special frequency in the cervical glands and in those at the root of the lung, more rarely in the abdominal glands; it constitutes the anatomical substratum of latent glandular tuberculosis, which clinically is largely comprised under the collective term "scrofulosis." Tuberculosis of the cervical lymphatic glands of children and youths develops with or without preceding primary affection of the peripheral organs;

for instance, eczema capitis, otitis, rhinitis, conjunctivitis, keratitis, blepharitis, pharyngitis—all of them chronic inflammatory processes which are characterized by rebelliousness, long duration, and marked tendency to relapse. Such primary affections may also be altogether absent, just as cheesy tuberculosis of the bronchial glands in children and youths, whose pulmonary parenchyma is perfectly normal, is often met with. In similar cases the tuberculous virus may pass through the mucosa or the lung tissue without leaving a trace, but, finding in the parenchyma of the lymphatic glands, conditions favorable to its increase it produces a primary glandular tuberculosis (cryptogenic infection). While on the one hand the gland takes the part of a filter, on the other hand the tuberculous virus extends by preference to neighboring glands; owing to the blocking and occlusion of normal lymph channels there results frequently also a retrograde (paradoxical) centrifugal transportion of the tuberculous virus, and thus are formed, as it were, cheesy bundles of glands the size of a hen's egg and larger. Latent glandular tuberculosis, especially about the root of the lung and in the mediastinum, is extremely common in children between the first and sixth year of life dead of some other disease. By a regionary spread from such glands secondary miliary tuberculosis of neighboring organs often results; for instance, we sometimes observe extension to the lungs from the intrathoracic glands (retrograde spread of the virus), or to the pleuræ or the pericardium; also from the mesenteric and retroperitoneal glands to the peritoneum. Besides, erosion of vessels (pulmonary veins) may cause the penetration of tuberculous cheesy glandular foci into the blood circulation, *i.e.*, hæmatogeneous auto-infection with the development of general miliary tuberculosis or of a metastatic tuberculous basilar meningitis.

Etiologically, glandular tuberculosis of children (tuberculous scrofulosis) is to be traced to intestinal infection by the food (the milk of tuberculous cows) and the accidental swallowing of infected material while crawling on the floor or to infection by the air.

Altogether different in their causation from this form of glandular tuberculosis, which is frequent in the children of tuberculous parents, are certain forms of subacute, subchronic, and chronic glandular swelling (benign, non-tuberculous form of scrofulosis) which, while occurring in subjects of the same age, has no connection with tuberculosis and whose course and termination (in the worst cases suppuration, abscess formation, and external opening) are as a rule favorable.

Secondary tuberculosis of the lymphatic glands is frequent in children and young persons, but much rarer in tuberculosis of older subjects. Anatomically it resembles primary glandular tuberculosis; after a certain length of time the glands are always enlarged and contain more or less scattered cheesy foci. Both primary and secondary glandular tuberculosis may terminate in spontaneous recovery by calcification of the caseous masses. In children the abdominal lymphatic glands, especially the mesenteric, retroperitoneal, portal, and epigastric, are sometimes enormously enlarged and cheesy (tabes mesenterica).

Neoplasms of the Lymphatic Glands.

As primary neoplasms we find lymphomata and sarcomata; the former are often with difficulty distinguished from simple hyperplasia (in leukæmia).

Malignant lymphomata is the term applied to tumors characterized by rapid growth and a certain malignancy; in their clinical course they belong to the lympho-sarcomata.

Chronic hyperplasia of the lymphatic glands without associated leukæmic disease of the blood is also largely described as pseudo-leukæmia (Hodgkin's disease), or when accompanied by grave anæmia as anæmia lymphatica; the relation of this affection to splenic anæmia is still rather obscure.

Besides round-celled lympho-sarcoma, primary fibro-sarcoma and spindle-celled sarcoma are occasionally observed.

Among the secondary and metastatic new formations of the lymphatic glands carcinoma occupies the first place. In cancerous disease of peripheral organs, cellular elements are almost regularly transported by way of the lymph channels into the nearest lymphatic glands, where they lodge and increase. The entire process is to be regarded as a kind of auto-transplantation, in which infectious germs may possibly play a part but are not absolutely essential. If we were to concede their activity from a theoretical point of view, they could be transported only within the cells, since metastatic carcinomatous disease of the lymphatic glands, like metastatic disease of other organs (lungs, liver, etc.), always permits the recognition of the same epithelial elements as those of the organ primarily affected. Cancerous lymphatic glands are always swollen, the cut surface is often medullary and whitish or grayish-white in color; sometimes we also find retrograde metamorphoses of the carcinomatous proliferations, such as fatty degeneration, softening, necrosis, and hemorrhage. As in tuberculosis, the lymphatic glands retard the spread of the cancerous disease, at least for a time. Owing to the blocking of the physiological channels, collateral and retrograde passages may develop and thus again give rise to metastases; in this manner, with uterine carcinoma, after occlusion of the retroperitoneal and other glands the inguinal or mesenteric lymphatic glands may be found in cancerous degeneration.

Very rarely, excepting in lympho-scaroma and melanotic sarcoma, we find metastatic sarcomatous disease of the lymphatic glands; metastasis in sarcoma is effected by preference through the blood-vessels.

DISEASES OF THE THYROID GLAND.

The thyroid gland is an organ which is indispensable to life, especially during the developmental period; it is a true gland, whose epithelia furnish a specific secretion (thyroidin) that is distributed through the lymph channels. When formed in excess or when its efflux is impeded, the secretion accumulates in the gland follicles in the shape of colloid masses.

Extirpation of the thyroid gland is borne by some animals, it is said, without danger to life, while dogs succumb. In man, after complete removal of the gland and after cessation of its function, cretinoid degeneration, myxœdema, and cachexia strumipriva

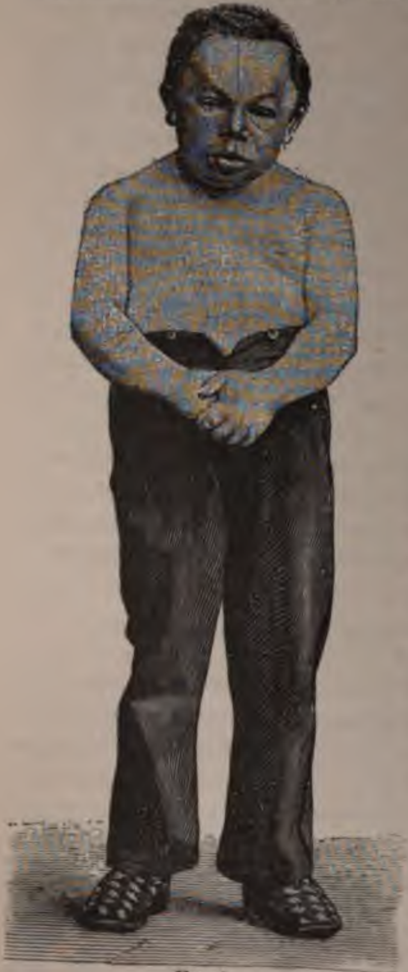

FIG. 4.

FIG. 4.—Cachexia Strumipriva.*

Patient, aged 28 years; height, 127 cm. = 51 in.; body small and boyish, but the size of the head and expression of the face correspond to the age. Kyphotic curvature of the upper dorsal spine. Features stupid, chiefly on account of the marked puffiness of the face, the thick lips, and the pale tint of the face and of the visible mucous membranes. External integument dry, here and there desquamating, having the peculiar soft feel of myxœdema. Scalp hair very scanty, no trace of a beard, pubic hair sparse. Speech slow and laborious, partly because of the enlargement of the tongue. Radial pulse barely perceptible. Abdomen abnormally large. Although the gross muscular power on the whole is preserved, the patient can hardly walk and is no longer able to perform the light work of knitting which a few years previously he could do skilfully.

Mental functions undeveloped, about like those of a boy six to seven years old. Death ensued rapidly after a relapsing attack of unconsciousness and dyspnœa. The autopsy showed complete absence of the thyroid gland, chronic leptomeningitis with moderate implication of the cerebral cortex, and negative results otherwise.

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*After R. Grundler: "Zur Kachexia strumipriva." Mittheil. aus der chir. Klinik zu Tübingen, 3 Hft., 1884, p. 200, pl. vi.

The patient had suffered from his eighth year with a thickening of the neck due to a goitre the size of a fist, which caused great dyspnœa and dysphagia. In his twelfth year—sixteen years before the fatal termination—the goitre was completely removed. As early as six months after the operation the relations of the patient observed a diminution of his mental activity; the boy, formerly bright and cheerful, became quiet, moody, and indifferent. Aside from the change in disposition it became soon evident that the trunk and extremities of the patient had ceased to grow, while the development of the head proceeded normally. On leaving school (at the age of fourteen) the patient was useless for a trade or for labor in the fields, and had to pass his time in knitting. Besides, a decreased acuity of hearing and vision was notable.

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result; when the gland is extirpated from young animals (lambs and kids) the sequelæ are dwarfed growth and cretinoid degeneration.*

The symptoms of cachexia strumipriva (Fig. 4) begin very gradually several months after the patients from whom the gland has been removed by operation have been discharged and the wound has healed. The preservation of small remnants of the gland, or the presence of accessory glands occurring in more than half of the cases, suffices to prevent the cachexia.

With reference to the normal relations of the thyroid gland it may be remarked that it rapidly increases at the age between eleven and twenty years (the period of puberty), about trebling its size (from 12–36 gm.=185–555 gr.). In men the normal thyroid gland (34 gm.=509 gr.) is somewhat larger than in women (30 gm.=463 gr.), probably in harmony with the greater body weight of the former. The average weight in adults ranges between 36 and 50 gm.=555 and 771 gr. Inflammation of the normal (thyroiditis) or of the enlarged gland (strumitis) is not frequent; the infectious form sometimes occurs in connection with typhoid fever and diphtheria.

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* The recent remarkable results of thyroid feeding in patients whose thyroid gland is absent or destroyed by degenerative processes show that the normal gland produces a specific substance (thyroidin) which is necessary to the preservation of health and of fundamental importance especially for the proper performance of the cerebral functions.

ENLARGEMENT OF THE THYROID GLAND. STRUMA OR GOITRE.

An enlarged thyroid gland is very frequently found in persons after puberty. In subjects between the eleventh and fiftieth years, dead of various diseases in Munich, the thyroid gland proved to be enlarged in nearly one-fourth (twenty-three per cent); in those over fifty, in more than one-half (fifty-six per cent). Women suffer from goitre more frequently than men (influence of pregnancy?).

The majority of observers include every enlargement of the thyroid gland among the neoplasms under the head of adenoma—a view which seems to be based neither on histology nor on physiology; most of the enlarged thyroid glands are to be classed as true hypertrophies or hyperplasiæ.

In hypertrophy of the thyroid gland (struma parenchymatosa sive hyperplastica) the organ appears uniformly enlarged, as much as double and treble the normal; in color, consistence, and minute structure it corresponds exactly with the normal gland. The enlargement develops slowly, and when moderate causes hardly any disturbance. This group includes congenital goitre, which is observed now and then in newly born children, the lateral lobes of the gland attaining the size of a walnut and larger.

Diffuse (interacinous) adenoma of the thyroid gland is characterized by abundant formation and deposition of colloid material and by round-cell infiltration of the supporting tissue. When in this alteration of the glandular substance there is an accumulation of larger colloid masses in the form of glossy, gelatinous spheres and drops, the condition is known as colloid or gelatinous struma; either the entire gland or some of its parts may be thus affected. At the same time there is frequently a tendency to the formation of cysts (cystadenoma, struma cystica); the gland follicles filled with colloid masses becoming dilated into cysts containing not only colloid and gelatinous but also, when large, a sanguineous fluid of a dirty brownish-red color (struma cystica hæmorrhagica). The cyst wall, which is sometimes identical with the capsule of the gland, is thickened, often hard, stiff, and partly calcified.

With abundant development of vessels, which even in the normal gland are characterized by their size and calibre, the variety known as struma vasculosa results; with predominance of the arteries, struma aneurysmatica; with that of the veins, struma varicosa.

When marked proliferation of the interstitial connective tissue is chiefly at fault in the enlargement of the gland, struma fibrosa or fibrous goitre results; the soft glandular parenchyma with a moist lustre is replaced by a firm fibrous mass. The deposition of lime salts in the older effusions of blood or in the indurated fibrous parts gives rise to struma calculosa.

All these various forms may be combined in different ways; thus we may find fibrous follicular forms, cystic goitre with calcareous deposits, effusions of blood—usually degenerative forms of goitre.

The adenoid neoplastic nature of goitre is besides clearly manifested by those forms which are known as nodular and are characterized by the fact that the enlarged gland contains circumscribed round nodules of different size (from a pea to a cherry and even half a hen's egg) and lighter color, consisting of embryonal glandular tissue. Such glands are most frequently found in young subjects.

When of long standing, the various forms of goitre often attain a large size, that of a fist and even a child's head. In such a case they exert great pressure upon surrounding organs, especially the trachea, which is narrowed, thus impeding respiration and causing a tendency to dyspnœa. Lateral compression of the trachea gives rise to the so-called sword-sheath form of the latter.

When the growth is downward, the strumous masses penetrate behind the manubrium sterni (struma substernalis), where the room is limited, so that the lower segments of the trachea are seriously compressed and grave disturbances arise. Sudden death, usually by arrest of respiration, is not unusual in the larger forms of goitre.

The enlargement of the thyroid gland is at times associated with hypertrophy of the heart and exophthalmus (exophthalmic goitre); this peculiar symptom complex, which is combined with numerous other, especially nervous disturbances, is also known as Basedow's or Graves' disease.

In some parts of the world goitre occurs endemically with great frequency. In its degenerative forms (fibrocystic goitre) it is often associated with cretinism, stunted growth, and pronounced physical and mental defects. The same condition also develops within a few years after complete extirpation of the thyroid gland in young subjects, in the form of cachexia strumipriva (Fig. 4).

Aside from senile atrophy, which is rare, we occasionally observe atrophy of the thyroid gland due to unknown causes; it is followed by a similar condition as in cachexia strumipriva (myxœdema atrophicum).

Malignant new formations (struma maligna) develop with special frequency in the enlarged thyroid gland; carcinoma more rarely than round-celled sarcoma. Within a few months the gland attains a large size and by pressure and constriction of the trachea, larynx, and œsophagus terminates fatally.

Diseases of the Respiratory Organs.

1. DISEASES OF THE NOSE.

Hemorrhages from the nasal mucosa occur frequently in connection with ulcerative and other inflammations, as a result of injuries, neoplasms, the hemorrhagic diathesis, and infectious diseases (typhoid fever, influenza).

Inflammatory processes of all sorts are often observed upon the nasal mucosa, more frequently in children than in adults; acute, subacute, and chronic catarrhs with chiefly sero-mucous or purulent secretion. Acute infectious catarrhs in particular have a tendency to extend downward into the pharynx, larynx, trachea, and bronchi.

Secondary forms of nasal suppuration are sometimes observed in connection with purulent inflammation of the accessory cavities or with caries of the cribriform bones.

Among the serious forms of rhinitis are erysipelatous and phlegmonous inflammations of the mucous membrane; they develop from erosions and ulcerations and extend by preference to the cutis and subcutis of the face and head.

Ozæna (rhinitis atrophicans fœtida) is characterized by a secretion which rapidly dries into crusts and furnishes a good culture medium for microbes, especially those of decomposition (mixed infection);

owing to their great increase fetor results and the mucous membrane atrophies in consequence of connective-tissue contraction. The process is observed chiefly in scrofulous and dyscrasic, more rarely in healthy subjects; occasionally it spreads to the nasopharynx and the accessory cavities of the nose.

Tuberculosis of the nasal mucosa is rare in its ulcerative form; it is secondary and differs in no way from tuberculous affections of other mucous membranes. The ulcers are usually irregularly round and have an eroded bottom; a rarefying osteitis of the adjoining bones, together with necrosis and the development of the micro-organisms of decomposition may be superadded.

Diphtheria of the nasal mucosa is observed at times secondarily in diphtheria of the pharynx; this disease is localized pre-eminently in the nasopharynx, the anterior half of the nose being in a condition of purulent inflammation, which involves also the maxillary cavities and the cells of the cribriform plate of the ethmoid bone. Owing to the associated enlargement of the pharyngeal tonsil, the swelling and exudation often cause considerable stenosis and occlusion of the nasal canal. In some infectious diseases (scarlatina, measles, and typhoid fever) there may be a similar affection as in the pharynx and larynx, in the shape of a necrosing (diphtheroid) rhinitis, which probably is invariably due to infection with streptococci.

In syphilitic ozæna we observe, along with catarrhal inflammation of the mucosa, ulcerative and destructive processes which spring from the originally cellular and gummous infiltration of the mucous membrane, of the perichondrium, or of the periosteum.

On the septum ulcerations are met with in the form of furrows passing in a longitudinal direction. The process extends by preference to the bones of the septum, especially the turbinated bodies; owing to the accompanying rarefying osteitis the affected bones acquire an almost wormeaten appearance, the bridge of the nose and sometimes the entire bony framework of the nose is depressed. In some cases there are in addition ulceration and destruction of the external soft parts, with defects in the tissues around the nostrils; perforation into the oral cavity after destruction of the soft and hard palate, *i.e.*, cleft palate, may be observed occasionally. In syphilis of the nose similar affections of the mucous membrane of the oro-pharynx are frequently found.

Neoplasms of the nasal mucosa are in the large majority of cases of the connective-tissue type; polypoid adenomata are more rarely observed. Polypoid fibromata are usually very vascular (angio-fibromata), or very soft, juicy, œdematous, and with a gelatinous lustre, *i.e.*, myxo-fibromata. Now and then they are multiple and represent transitions between polypoid rhinitis and true neoplasms. These tumors, briefly described as nasal polpyi, are attached particularly to the extremities of the middle turbinated, to the roof and the external wall of the nose, and in the furrow between the inferior and middle turbinated bodies. In some cases the surface is more uneven and raspberry-like.

Hard fibroma, fibro-sarcoma, sarcoma, and carcinoma are rare; the latter sometimes extends from the external skin to the nose.

2. DISEASES OF THE LARYNX.

The inflammatory processes of the larynx coming under observation are rarely primary but more often secondary. Inflammations of the nasal and pharyngeal mucosa extend by contiguity to the larynx and trachea (*e.g.*, descending croup), and on the other hand inflammatory processes from the bronchi and trachea are propagated into the larynx (ascending processes). Besides, catarrhal laryngitis occurs symptomatically in acute infectious diseases such as measles, whooping-cough, typhoid fever, and small-pox.

The anatomical alterations in acute and chronic catarrhal laryngitis resemble those of inflammation of the mucous membranes in general.

Chronic laryngeal catarrh occurs especially in drunkards, in connection with pulmonary tuberculosis, and as a result of habitual overexertion. A special variety of chronic inflammation is hyperplastic laryngitis, which causes papillary proliferations (pachydermia verrucosa), particularly on the mucous membrane between the arytenoid cartilages and at the posterior end of the vocal cords, where larger papillæ are normally present.

Diphtheritic and Croupous Laryngitis.—Croup and diphtheria of the larynx are found most frequently in idiopathic diphtheria of the pharynx. The croupous inflammation (Plate 19) causes the formation of a yellowish-gray or whitish false membrane, which covers either the inner surface of the whole larynx or only some of its parts; the tubular membrane often extends to the trachea and the larger bronchi. The mucous membrane is swollen, injected, and sometimes contains scattered hemorrhages; the epithelium is necrotic. In other cases we observe along with the formation of croupous deposits spots of a dull gray infiltration of the mucous membrane, especially on the vocal cords; also superficial and deep eschar formation which terminates in ulceration.

Diphtheroid laryngitis with final ulceration is observed in about twelve per cent of all cases of typhoid fever; the ulcers sometimes extend down to the cartilage with resulting perichondritis; usually they occur in the third week of the disease.

Acute erysipelatous inflammation of the mucous membrane (Plate 21) and submucosa occurs now and then primarily in persons in previous good health. This dangerous affection, which may cause sudden death by stenosis of the larynx, is largely and rather loosely described as acute œdema of the glottis. Secondarily we meet with inflammatory œdema of the larynx and the aditus (aryepiglottic folds) in numerous inflammatory and ulcerative processes of the same organ and of the fauces, in angina Ludovici, in prevertebral abscesses, etc. In rare cases we observe phlegmonous laryngitis with extensive purulent infiltration and great swelling of the mucosa and submucosa, now and then in connection with a similar affection of the pharynx (pharyngo-laryngitis).

Ulcerative laryngitis is occasionally found as a sequel of chronic catarrh or associated with it; the ulcers are shallow, superficial, and more like erosions.

Ulcerative tuberculous laryngitis (Plate 20) occurs in about one-fifth of all cases of chronic pulmonary tuberculosis. The ulcers begin as a rule in the region of the posterior point of attachment of the vocal cords and gradually extend downward; after the perichondrium is destroyed the cartilage may become necrosed. The ulcers look eroded and pale; at the bottom and the margins yellowish-white nodules are sometimes visible; the margins are usually markedly tumid and consist of proliferated epithelial masses. Recent ulcers are shallow, the older lesions deeper and excavated. The healing of such ulcers is certainly quite rare. Tuberculosis of the larynx arises by way of intrabronchial and intratracheal auto-infection, from transportation of the infectious bronchial and pulmonary secretion; the stagnation of portions of the sputa in the depressions of the larynx favors the local infection. The rarity of laryngeal tuberculosis in children probably depends on the lesser frequency of cavities in the lungs and the more rapid course of the disease in young subjects.

Syphilis of the larynx occurs either in diffuse patches of infiltration of the mucous membrane (mucous patches) or in the form of excavated ulcers, located usually on the vocal cords, at the posterior wall of the larynx, and on the margins of the epiglottis. The ulcers have a lardaceous, yellowish-white bottom and tumid margins; along with them we occasionally find flat nodular gummata, irregular cicatrices, purulent perichondritis, and necrosis of the cartilage.

NEOPLASMS OF THE LARYNX.

The neoplasms of the larynx (polypi) belong in the large majority of cases among the connective-tissue tumors. Their sites of predilection are the true vocal cords and their anterior commissure. The first development can often be traced to antecedent inflammations and local erosions such as form on a catarrhal basis. The tumors here in question represent most frequently papillary fibromata and epitheliomata, which constitute more than one-half, almost two-thirds of all laryngeal tumors. Their surface is verrucose, nodular, or villous, corresponding to the papillary hyperplasia of the superficial layers of the mucosa. A second group of laryngeal tumors is formed by the nodular fibromata (fibroma tuberosum), which constitute about one-fourth to one-third of all polypi; they spring from the deeper connective-tissue layers of the mucosa and are seated chiefly on the true vocal cords. The rest of the polypi, about one-tenth of all cases, are simply mucous polypi and cysts (mxyo-fibroma and adenocysto-fibroma).

Malignant tumors (sarcoma and carcinoma) are very rare as primary formations in the larynx; carcinoma sometimes develops from the papillary epithelioma when of long standing. Secondarily and by extension from the surrounding structures laryngeal carcinoma occurs occasionally when the primary focus of disease is in the pharynx, the base of the tongue, the œsophagus, and the thyroid gland.

TRACHEA AND BRONCHI.

The trachea usually participates in the various diseases of the larynx and the bronchi; especially in inflammatory processes the larynx and trachea are largely affected coincidently—for instance, in croupous laryngo-tracheitis in connection with diphtheria of the pharynx.

Erosion and necrosis of the trachea are now and then caused by an aneurism of the thoracic aorta, also by cancer of the œsophagus; in the former case the result is a sudden hemorrhage from the point of rupture of the aneurism; in the latter case, fetid bronchitis and inhalation pneumonia.

Injuries of the trachea are usually caused by operation (tracheotomy) or are of accidental origin, *e.g.*, in attempted suicide when incised wounds open the trachea. When large vessels are injured at the same time, large masses of blood enter the trachea and may cause asphyxia, and when the œsophagus is also wounded food particles may pass into it. Under normal conditions the smaller tracheal wounds heal by the formation of a linear connective-tissue cicatrix; sometimes a fistula remains behind.

Stenoses and deformities of the trachea result most frequently by compression from without, *e.g.*, by the formation of abscesses in the neighborhood, by aneurisms, and in the majority of cases by enlargement of the thyroid gland, which causes the sword-sheath form of stenosis of the trachea (Figs. 5–15); also atrophy of the cartilaginous rings, or their connective-tissue metamorphosis. Besides, the lumen of the trachea is occasionally diminished and occluded by inhaled foreign bodies, more rarely by syphilitic cicatrices and infiltrations which cause strictures situated at times above the bifurcation.

Tuberculous ulcers in the trachea are of comparatively rare occurrence; sometimes they are extensions from the mucosa of the larynx. They correspond in the main with the alterations in the larynx or form multiple, closely crowded, lenticular defects in the

Figs. 5-15.—Strumous Stenoses of the Trachea.*

Fig. 5.—Sword-sheath form caused by simple enlargement of both lobes of the thyroid gland, the trachea being in median position.

Fig. 6.—Unilateral compression of the trachea by goitre of one side.

Fig. 7.—Spirally curved sword-sheath form caused by compression of the trachea from both sides but at different levels. Goitre of moderate size.

Fig. 8.—Spirally curved sword-sheath form; compression by a very large right-sided goitre with simultaneous marked backward development, thus compressing the trachea from the right and behind, while a smaller lobe farther down presses upon the trachea from the left anteriorly.

Fig. 9.—Spirally curved sword-sheath form; a bilateral retro-œsophageal large goitre, irregular prominences of which, directed toward the trachea, have produced the spiral form of the latter.

Fig. 10.—True sword-sheath form, produced by uniform enlargement of both lobes of the thyroid gland, the trachea running a median course.

Fig. 11.—Sword-sheath form; produced as in Fig. 6.

Fig. 12.—Arc-like curved sword-sheath form; both lobes of the thyroid being nearly equally hypertrophied, but the right lobe had developed more deeply toward the trachea.

Fig. 13.—Irregular compression of the trachea by an enormous carcinoma of the thyroid gland (struma maligna).

Fig. 14.—Double compression of the trachea; in the upper part, from the cricoid cartilage down, from in front, both lobes and the isthmus were much enlarged, the right lobe extending far forward and causing the upper compression; farther down the trachea appears bent in from the left by the left lobe of the gland.

Fig. 15.—Sword-sheath form, unilateral compression; the right lobe of the thyroid gland the size of a fist, the left that of a walnut.

mucous membrane, which is at times markedly injected and diffusely inflamed.

While the larger bronchi normally and pathologically resemble the trachea, the smaller and thin-walled bronchioles participate in most of the affections of the pulmonary tissue.

Inflammation of the bronchial mucosa (bronchitis) occurs as a primary and idiopathic affection or secondarily as a sequel and concomitant of pulmonary maladies, in acute infectious diseases; also in connec-

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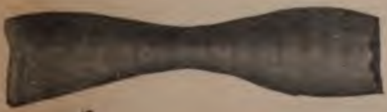
*After E. Müller (Tübingen): "Mittheilungen aus der chir. Klinik zu Tübingen," by Paul Bruns, 3 Heft, Tübingen, 1884.



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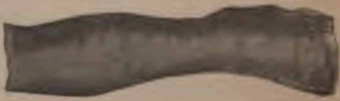

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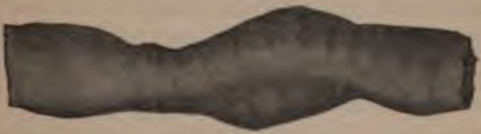

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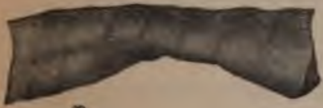

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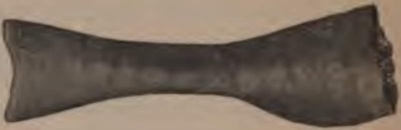

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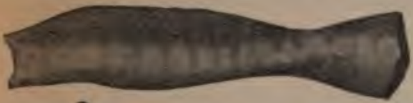

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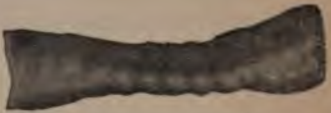

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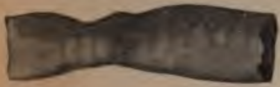

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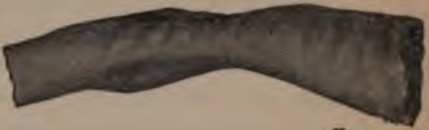

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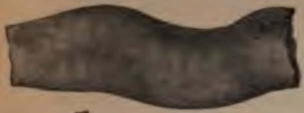

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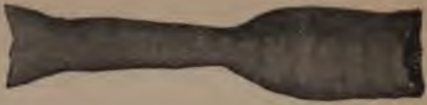

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tion with cardiac circulatory disturbances in numerous affections of the heart and its valves, in consequence of which a congestive hyperæmia develops, especially when the efflux into the left heart is impeded.

Acute bronchitis may be localized in the larger or smaller bronchi and is found in all possible gradations, from the milder forms beginning with seromucous secretion to the intense purulent variety with abundant, thick, creamy or greenish-yellow secretion (blennorrhœa). When expectoration has been insufficient, as in cases of debilitated patients, in advanced or very early age, the bronchi are found filled with yellowish-white purulent secretion; when, after the pulmonary parenchyma is divided and the cut surface is carefully scraped, light pressure is exerted upon the tissue, the medium-sized and smaller bronchi give exit to purulent plugs which are easily seen.

Chronic bronchitis is extremely frequent in disease of the pulmonary parenchyma (tuberculosis, emphysema) and of the heart (congestive bronchitis). The mucous membrane, which is generally swollen, is of a brownish-red color and the surface is covered with tough muco-purulent masses; when the process terminates in atrophy of the mucosa, secondary bronchiectasis sometimes follows.

Croupous or fibrinous bronchitis develops either downward in connection with the same inflammation of the trachea and larynx, or in the inverse direction, upward, in croupous pneumonia. In the lumen of the bronchi, whose mucosa is greatly swollen and reddened, are solid or tubular branching coagula of a pale yellowish or grayish-white color, which can often be drawn out entire if some care is exercised. The same disease occurs now and then in an apparently idiopathic, chronic, and relapsing form.

Diphtheritic necrosing bronchitis, with the formation of necrotic hemorrhagic foci, is very rarely observed in grave cases of pharyngeal diphtheria.

Fetid or putrid bronchitis develops either simultaneously with pulmonary gangrene or follows it, especially after the inhalation of foreign bodies liable to decompose (food particles). This variety also occurs secondarily in bronchiectasis, in which the conditions—the accumulation of bronchial secretion in the dilated bronchi—favor the deposition and propagation of the germs of decomposition. In the worst cases fetid bronchitis causes now and then a fatal broncho-pneumonia or else general septicæmia or septico-pyæmia.

Stenosis and occlusion of the bronchi (bronchostenosis) results in the smaller bronchi from inflammatory swelling of the mucosa or from the accumulation of pathological products such as pus, mucus, blood, serum (pulmonary œdema), and finally from the inhalation of foreign bodies, such as shirt buttons, cherry pits, fragments of bone, etc. Foreign bodies penetrate more frequently into the right than into the left bronchus, because the former passes in a direction more nearly vertical and constitutes more of a direct continuation of the trachea. When the foreign body is freely movable or but loosely impacted, there is danger that a fit of coughing may propel it into the glottis with death from asphyxia at a late period. Foreign-body bronchitis may lead to pneumonic processes that may terminate fatally after a long time, now and then only after decades. The stenosis of the bronchi, especially those devoid of cartilage, may also be due to pressure (compression of the lung by a pleuritic exudate or by hydrothorax), to the effect of an aortic aneurism or of lymphatic glands which have become abnormally enlarged by carcinomatous, sarcomatous, or tuberculous disease.

DILATATION OF THE BRONCHI (BRONCHIECTASIS).

(Plate 22.)

The dilatation of the bronchi is either diffuse, when the bronchial tube is found uniformly dilated for considerable distances, *i.e.*, cylindrical bronchiectasis; or the dilatations are more circumscribed, fusiform, sacculated, or rosary-like, with intervening constrictions. True bronchiectasis is invested with a mucous membrane having normal ciliated epithelium; false bronchiectasis is the condition in which the spaces connected with the lumen of the bronchi are only partly covered with ciliated mucous membrane, while another portion corresponds to real cavities resulting from necrotic disintegration of the pulmonary parenchyma. The causes of bronchiectasis are manifold and often hard to determine from the anatomical findings. In general, chronic changes in the walls with alteration of the vital qualities (elasticity and contractility, atrophy of the muscular coat) following upon chronic inflammatory processes play the principal rôle (nutritive disturbances); next in order are mechanical influences, the air pressure being greatly increased at frequent intervals; finally dilatation may be due to contraction and condensation of the surrounding pulmonary parenchyma (hypertrophic bronchiectasis, Plate 22), to the traction exerted by the gradually expanding pulmonary tissue during the absorption of a pleuritic exudate upon the peribronchial connective tissue, upon the connective-tissue sheath of the bronchus. Besides, accumulated exudates obviously favor the dilatation of the bronchi, while the distention in turn of course hinders the prompt evacuation of the secretion and favors the accumulation of muco-purulent inflammatory products. In the majority of cases, particularly those of which primary changes of the walls form the main cause, the wall, especially the cartilaginous and glandular portion of the dilated bronchi, is atrophic (atrophic bronchiectasis). With reference to the localization it may be remarked that the inferior lobe is more frequently the seat of bronchiectasis than are the upper and middle lobes; at the apex of the lung bronchiectases of slight extent (from the size of a pea to that of a cherry) are more frequent in connection with indurated and cicatricial processes (apical tuberculosis). Bronchiectasis is usually associated with intractable chronic bronchitis, and occasionally we observe the development of a fetid bronchitis with a tendency to fatal general infection (septicæmia).

DISEASES OF THE LUNG.

Among the circulatory disturbances of the lung, one of the most frequent and important is venous or *congestive hyperæmia* (Plate 23). This develops when the efflux of the blood from the pulmonary veins into the left auricle is interfered with (in valvular defects of the left heart and in heart failure), and leads first of all to a dilatation of the capillary network (capillary ectasia). The capillaries are markedly tortuous, resembling varices, and project deeply into the alveoli; at the same time there are a condensation of the pulmonary framework and widening of the septa. The increased capillary pressure causes continual diapedesis of red blood corpuscles, which change into granular and liquid hæmatoidin. The latter is deposited in the pulmonary stroma and particularly in the alveolar epithelia and leucocytes. The latter are sometimes called heart-lesion cells, because their occurrence in the septa indicates lesions of the heart and pulmonary congestion of cardiac origin. Owing to the enormous accumulation of the rusty brown coloring matter in the pulmonary parenchyma and the associated fibrillary hyperplasia of the stroma of the lung, alterations develop which are known as "brown induration." In the higher grades of the condition the lung is of a rusty color, in the initial stages more reddish-brown, and of a firm consistence; as a rule the bronchi are in a state of chronic catarrh (congestive catarrh).

Another form of venous hyperæmia is the hypostatic variety which is often met with in the posterior and inferior segments of the lung in cases of cardiac weakness, of prolonged agony, and of permanent dorsal position of the patients. In this vital hypostasis the affected portions of the lung appear overfilled with blood, of a dark brownish-red color, waterlogged, and the contained air is correspondingly diminished; the passage of the blood into the air spaces of the lung and thence into the bronchi is often observed. This hypostatic hyperæmia occasionally results in pneumonia (hypostatic pneumonia), when the tissue exhibits a spleen-like consistence. Post-mortem hypostasis, a cadaveric phenomenon, shows the above-described changes in a minor degree; this form is found in a most characteristic type in subjects whose death was sudden and when a prolonged period of time intervened between the death and the post-mortem examination.

PULMONARY HEMORRHAGE.

Hemorrhages into the pulmonary tissue may be of traumatic origin, such as stab and gunshot wounds or extraneous blunt force acting upon the thorax; when the pulmonary pleura is injured at the same time the blood is effused into the pleural sac (hæmothorax), and may also enter the bronchi, where it is mixed with air, becoming frothy and bright red. Effusions of blood into the lung occur also from the erosion of larger vessels in ulcerative and necrosing inflammations of the organ, *e.g.*, pulmonary tuberculosis and gangrenous processes. Blood which reaches the smaller bronchioles and pulmonary alveoli by way of aspiration from the upper air passages or from a cavity usually causes multiple dark brownish-red foci which are not sharply demarcated from the surrounding tissue. Non-infectious pure blood penetrating into healthy lung in the above-described manner causes desquamation of the alveolar epithelium, of the epithelium of the finest bronchioles, also an immigration of leucocytes, sometimes secondary peribronchitis, a proliferation of the interstitial connective tissue, and a kind of desquamative pneumonia.

When infected blood is effused into healthy or diseased portions of lung, various forms of inhalation pneumonia result.

A special group is formed by hemorrhagic infarctions produced by embolism of branches of the pulmonary artery. These infarctions vary in size from that of a pea to that of a walnut, but sometimes they attain the size of a fist, so that they occupy the major portion of a lobe. Their shape is that of a wedge, whose point corresponds to the occluded arterial branch, the base to the pulmonary pleura. The infarctions, moreover, project markedly beyond the surrounding tissue, are firm in consistence, devoid of air, and blackish-red in color; the overlying pleura is generally covered with a delicate network of fibrin. Not every embolic occlusion of the pulmonary arterial twigs causes the development of a hemorrhagic infarction; in cases of occlusion of the main trunks of the pulmonary artery and in extreme anæmia and weakness of the heart there is often absolutely no reaction in the affected vascular region. Aside from arterial occlusion of sudden origin, the occurrence of hemorrhage, especially in the pulmonary capillaries and veins, is favored by increased cardiac blood pressure, such as exists in valvular disease of the left heart, in degenerative conditions and exhaustion of the heart muscle. This is the explanation of the fact that hemorrhagic embolic processes are observed with special frequency in a lung that is in a condition of chronic congestion, of brown induration. As a rule it is possible to demonstrate the primary thrombus which forms the starting-point of the embolism in the right ventricle or auricle or in the distribution of the peripheral veins. When a primary thrombus cannot be found we must remember that it is possible that an autochthonous marasmic thrombosis in the course of the branches of the pulmonary artery may likewise cause the development of infarctions; finally the primary thrombus may also have been completely swept away without leaving a trace.

When the embolus is infectious, it acts not only mechanically, interfering with the circulation, but it causes along the distribution of the occluded artery a circumscribed purulent inflammation, the formation of metastatic abscesses, which are usually multiple and subpleural in their location and give rise to secondary purulent pleurisy. Such pulmonary abscesses constitute the anatomical substratum of embolic septico-pyæmia, such as may develop, for instance, in connection with infected wounds or with septic puerperal metrophlebitis and terminate fatally.

Œdema of the Lung.

Œdema represents a very frequent anomaly of the pulmonary tissue and occurs in various forms and gradations, either distributed uniformly over both lungs or confined to isolated regions, especially the posterior and inferior portions of the lung after the horizontal dorsal position has been long maintained by the patient. The tissue appears more or less water-logged, with corresponding diminution of the contained air, the weight is increased, the consistence in the higher grades is doughy and almost resembling that of the spleen, the volume is augmented; the contained blood varies—in œdema of very rapid development it is usually increased, in forms of slow onset it is reduced and sometimes almost *nil.* In the latter case, in œdema of slow development (chronic œdema), the fluid escaping from the cut surface very abundantly on slight pressure is not frothy but contains only sparse air bubbles. In the most pronounced cases observed in valvular disease of the left heart and after prolonged agony, in which states transitional to desquamative pneumonia develop by reason of excessive proliferation and shedding of the alveolar epithelium, the œdematous tissue is very pale, frail, brittle, in a condition of gelatinous softening (pneumomalacia)—a state which sometimes merges into gangrene in consequence of the extreme disturbance of nutrition and the lodgement of germs of decomposition. The bronchi and trachea contain in pulmonary œdema a frothy, sometimes sanguineous matter (hemorrhagic œdema). Œdema of the lung is due to various causes—in the form of collateral œdema it progresses *pari passu* with collateral hyperæmia; in the neighborhood of inflammatory infiltrations it belongs in part in the division of serous inflammations. In the form of cardiac or congestive œdema it develops in valvular defects and exhaustion of the left heart; in another group of cases the œdema, usually associated with hydrothorax, is a local phenomenon of general dropsy (*e.g.*, in nephritis), or may be traceable to an alteration of the vessel walls. A decided influence of congestion and impaired circulation upon the development of œdema is shown most clearly by the frequent partial œdema in the posterior segments of the superior and inferior lobes following the horizontal position of the patient (hypostatic œdema).

EMPHYSEMA OF THE LUNG.

Pulmonary emphysema presents two principal forms—alveolar and interstitial emphysema.

Vesicular emphysema of the lung, a chronic organic affection of the organ, is to be distinguished from acute inflation of the lung; the latter represents a functional transitory dilatation of the alveoli (alveolar ectasia) and generally results from increased inspiration and impeded expiration, so that more air is inhaled than can escape during expiration. This acute pulmonary inflation (vicarious emphysema) occurs in numerous acute diseases of the lung, especially in children, for instance, in capillary bronchitis, pneumonia, and atelectasis; it shows itself very characteristically at the median margin of the lung (marginal emphysema).

In vesicular emphysema the lungs appear enlarged and cover the pericardium almost completely. The lungs do not collapse, have a puffy feel, retain the impression of the finger, and are diminished in weight. The enlarged pulmonary vesicles are visible through the pleura; on close inspection it is seen that the larger spaces do not consist simply of dilated alveoli but that they have resulted from the confluence of adjoining alveoli caused by atrophy of the septa.

The condition may be due to various causes.

Mechanical emphysema, so called, is produced by oft-repeated, prolonged, excessive inspiration and prolonged expiration. The continuous increased intra-alveolar pressure causes greater tension of the alveolar septa and their compression from two sides; the associated tension of the vascular network produces stretching and narrowing of the vessels and impeded circulation. As a result of the excessive tension and traction upon the meshes of the vessels the latter are so much narrowed that they finally admit the passage of serum only, which is insufficient for the nutrition of the vascular tissue. The capillary network becomes partly obliterated, the epithelium undergoes fatty change, the elastic elements give way and disappear; in this manner is produced a partial erosion of the septa, the development of defects which enlarge steadily until at last two or more septa coalesce. The thinning of the septa proceeds progressively with the increase in circumference of the alveoli. This rarefaction and atrophy of the pulmonary framework, which is started by the obliteration of vessels, is bound to develop more rapidly and more easily in ill-nourished and young subjects in whom the nutritive and physical conditions of the lung present a greater predisposition. This applies also in part to senile emphysema, in which the primary atrophy of the pulmonary tissue without general enlargement of the volume of the lung plays the main part, and in which a normal or slightly increased pressure is liable to change the insufficiently nourished pulmonary tissue in a manner similar to that described above. The statements here made will explain why the pure form of mechanical emphysema—as it were a professional affection—is found by preference in persons who subject their lungs to excessive, prolonged, and oft-repeated strain, *e.g.*, in the playing of wind instruments, in long-continued loud speaking and screaming, in laborious muscular exertion, forced marches, and inordinate mountain climbing.

A long-standing bronchitis associated with impeded expiration favors the development of emphysema, and inversely the obliteration of the pulmonary capillaries causes a disposition to circulatory disturbances in the bronchial mucosa and a secondary bronchial catarrh.

Great importance attaches to the sequelæ of pulmonary emphysema upon the heart and the circulation. Owing to the obliteration of a portion of the pulmonary capillaries the blood is dammed back in the pulmonary arteries, the pressure in the right heart rises, and dilatation and compensatory hypertrophy of the right ventricle and auricle ensue; finally, when the compensation is insufficient and the right heart flags—a condition which may be the cause of death—general venous congestion results. As in cases of mitral stenosis we find evidences of chronic congestion, namely, nutmeg liver, cyanotic induration of the spleen and kidneys, congestive catarrh of the stomach and intestines, moderate dropsy; in a word, when a fatal termination results from pulmonary emphysema, the death is due to the heart, not to the lungs.

Interstitial or interlobular emphysema (Plate 24) is also produced by the tearing of the alveolar walls with the escape of air bubbles into the interlobular connective tissue of the lung. In the subpleural connective tissue and also upon the cut surface we observe the air bubbles—usually from a pin's head to a hempseed in size—arranged like a string of beads. In some cases the air may extend to the connective tissue at the root of the lung, to the mediastinum, and thence to the subcuticular tissue of the neck and the adjoining parts (subcutaneous emphysema). Interstitial emphysema develops as a terminal condition in consequence of intense dyspnœa, most easily in the delicate lungs of children, especially in diphtheria, bronchitis, and pneumonia.

ATELECTASIS.

By the term atelectasis is understood a defective expansion of the pulmonary parenchyma; it may be found spread diffusely over both lungs (fœtal atelectasis) or confined to single lobes or lobules.

Pulmonary tissue thus changed is sunken, discolored bluish-red or ashy gray, airless, hard to cut, and resembles splenic tissue; atelectatic portions of the lung can be inflated from the bronchi, while this is not possible with the inflammatorily infiltrated and airless lung. The following forms are distinguished:

Fœtal or congenital atelectasis. This affects the entire lung, and total atelectasis of the newly born is a sure sign that the child did not breathe after birth, *i.e.*, that it was dead-born. In inanition with defective function of the respiratory mechanism we find inflated light red portions side by side with atelectatic segments.

Acquired atelectasis results from various causes. Pressure from without acting upon the lung produces compression atelectasis, *e.g.*, when fluid or air accumulates in the pleural cavity (hydro-pyo-pneumothorax). In cases of moderate accumulations of exudate only the inferior and posterior portions of the lung become airless; when large amounts of fluid are effused into the pleural cavity, the lung, retracted toward the root, changes to a flat cake-like mass, barely the size of a hand, which is devoid of blood and air, tough like leather, of a lead gray or slate color. The more recent the atelectasis and the shorter its duration, the more easily air again enters and *restitutio ad integrum* follows; the longer the atelectasis has existed the more difficult becomes restoration to health, hence the early operative removal of the pleural exudate is urgently indicated. Pressure from without is also the cause of that form of atelectasis which occasionally develops about the anterior sharp margins of the lower lobes in abnormal and prolonged elevation of the diaphragm consecutive to excessive distention of the abdomen (tympanites, peritonitis).

Obstructive atelectasis results from occlusion of the bronchi in consequence of swelling of the mucosa and the accumulation of secretion. Corresponding to the arborization of the occluded peripheral bronchi we observe most frequently at the sharp margins of the lobes depressed lobular, bluish-red, and airless patches which may coalesce and gradually occupy the greater portion of a lobe. This form is most common in infantile bronchitis.

Symmetrical atelectases are occasionally found in the posterior and inferior lobes of children dead of pharyngeal diphtheria or whooping-cough. In these cases, besides the occlusion of the bronchi the changed respiratory mechanism may be at fault, since it leads to excessive expansion of the upper and compression of the lower segments of the lung.

DISEASES DUE TO THE INHALATION OF DUST.

Pneumonoconiosis.

(Plate 25.)

The air spaces of the lung continually take up particles of dust: a portion of this passes out again with the expired air and the bronchial secretion, and a portion is retained in the pulmonary tissue, being partly fixed in the parenchyma, partly transported farther along the lymph channels. With reference to the pathogenic influence of the several kinds of dust the following points are of importance: the quality and quantity of the dust, the duration of the daily period of inhalation, the fact whether the dust is inspired continuously or at intervals, and also the vulnerability of the lung and of the individual. Healthy lungs are better able to expel the dust again, while it is more easily and in larger amounts retained in diseased organs.

The dust particles, which can be demonstrated microscopically and chemically both in the pulmonary tissue and in the peribronchial glands, are taken up by the alveolar wall and carried along in the juice and lymph current, either free or inclosed in phagocytes. A portion is retained in the lungs, where it causes different pathological processes. The dust deposited in the pulmonary tissue is partly intracellular, partly free. While the alveolar walls as a rule show but little deposited dust, this is found more abundantly in the peri-infundibular, peribronchial, perivascular, and subpleural connective tissue.

The more important forms of pneumonoconiosis distinguished are:

1. *Anthracosis*, soot or carbon lung (Plate 25, Fig. *b*), the most frequent form, is found in numerous gradations in the majority of persons. In the initial stages the lungs appear blackish discolored, partly in spots, partly in meshes; the particles of carbon are very small, roundish, and almost regular in the form caused by soot, while the particles of coal and charcoal are more irregular in shape, jagged, angular, pointed, and frequently of considerable size (carbon or miner's lung).

The initial stages and slighter grades are called anthracosis simplex, which is to be differentiated from the marked forms characterized by greater alterations in the pulmonary parenchyma, known as indurative anthracosis, the typical coal-miner's lung, often combined with chalicosis (indurative anthraco-chalicosis). The accumulation of soot and carbon particles is usually greater under the pleura than in the parenchyma; the upper lobes are as a rule more markedly affected than the lower lobes, but the infiltration of the apex is no greater than that of the remainder of the upper lobes. The portion of both lobes situated close to the spinal column behind the hilus is generally also greatly infiltrated. The adjoining edges of the upper and lower lobes usually show an intensely black border; the surfaces that do not touch the costal pleura contain but little carbon in their subpleural portions. In the pulmonary parenchyma the distribution is rather uniform; indurated condensed portions and those surrounding cavities exhibit usually a more marked slaty discoloration.

2. *Chalicosis*, stone lung (Plate 25, Fig. *c*), is found in workmen laboring in an atmosphere charged with stone dust, especially in stone-cutters, millstone-dressers, glass and diamond cutters. To the same class belongs in the main also the inhalation of street dust. The mineral dust composed chiefly of silicates and limestone grit irritates the mucous membrane of the respiratory canal and the pulmonary tissue far more than do soot and coal dust. In advanced cases fibrous peribronchitis and perivasculitis, now and then also chronic broncho-pneumonia, result. When the injurious influence has been more prolonged and more intense, especially if caused by inhaled irregularly angular and pointed particles of quartz, a diffuse disease of the pulmonary tissue, indurative chalicosis, may develop. In pronounced cases we find the pleura as a rule as rough as a grater, owing to the numerous subpleural deposits of fibrous nodules, about the size of a hempseed, which can also be felt and seen on the cut surface.

3. *Siderosis*, metal-dust lung, exists in several varieties—the red-iron lung (Plate 25, Fig. *a*), when the red oxide of iron, such as is used in coloring blotting-paper, is inhaled in large amounts for a considerable length of time; the black-iron lung, when the ferroso-ferric oxide is inhaled. At first we find, as in chalicosis, small grayish translucent nodules, later firm fibrous nodules and tubercles which tend to coalesce and resemble in appearance the sole of a shoe closely studded with nails. Finally, in the advanced stages, a diffuse cirrhosis results, which is especially characteristic by its intense rust-colored markings after the inhalation of red oxide of iron.

The most dangerous effect upon the lung is evidently produced by the dust of the so-called Thomas slag, which is extremely irritating to the pulmonary tissue and tends especially to cause typical, infectious, croupous pneumonia.

A similar effect, though less serious, is produced by the habitual inhalation of vegetable dust by workers in tobacco, cotton, wooden ware, hemp, flax, etc.

All these substances, particularly if mixed with pathogenic germs, readily cause bronchitis and inflammatory states in the lung, so-called accessory processes, which often occur after the inhalation of mineral and metallic dust, when they assume the form of catarrhal and croupous pneumonia and especially accessory tuberculosis. The fact that with the characteristic soot or carbon lung tuberculosis is comparatively rarely met with is ascribed by some authors to the specific (antibacterial?) effect of coal; at all events the indurative and productive processes which occur in many conioses of the lung occlude numerous lymph and blood channels that otherwise would favor the spread and metastasis of the tuberculous virus.

On the other hand, the spread and progress of originally latent and localized tuberculous processes in the lung are favored by the inhalation of the various kinds of dust, while the healing and arrest of tuberculous processes are retarded.

INFLAMMATION OF THE LUNG (PNEUMONIA).

Catarrhal Pneumonia.

Lobular Pneumonia, Broncho-Pneumonia.

Catarrhal pneumonia progresses downward, cen
fugally, since the inflammatory process extends fr
the mucous membrane of the smaller bronchi (ca
lary bronchitis) to the corresponding lobules of
lung. The inflammation attacks by preference
inferior and posterior segments of the lung; in
aërated, light-red tissue we observe scattered lobu
foci which are sharply demarcated from the surrou
ing structures by their dark bluish-red color, th
firm and spleen-like consistence. The cut surface
not smooth but marked by flat prominences, since
infiltrated lobules, which contain no air or only tra
of it, are apt to project and differ from the surrou
ing tissue by their color, at first darker, then light
and the greater mass of blood they contain.
scraping the cut surface while slight pressure is
erted upon the tissue, plugs of creamy pus exude fr
the divided finer bronchi. In the further course
notice on the cut surface or through the pleura th
the central portions of the affected lobules in part
ular are somewhat lighter colored, sometimes speckl
with grayish-yellow spots, corresponding to the co
mencing purulent disintegration which may advan
to the formation of abscesses.

When the process is more extensive the lobul
foci may coalesce so that at last the greater porti
of a lobe or the entire lobe shows splenization (co

fluent lobular pneumonia). The pleura as a rule is not involved or exhibits at most the initial stages of a fibrinous inflammation. The exudation is mainly purulent, sometimes there are also small quantities of fibrin and alveolar epithelia in varying numbers in the alveoli of the diseased parts.

The pathological features are less characteristic and somewhat obliterated in the lower lobes when the process is associated with hypostasis and œdema (asthenic marasmic pneumonia); frequently there are also present lobular atelectases the result of occlusion of the bronchi. In the development of the disease various factors co-operate—imperfect expectoration of the bronchial secretion and its gravitation toward the deeper portions of the lung, some weakness of the respiratory mechanism, and reduced cardiac activity. This form of pneumonia therefore occurs by preference in children, in the aged, and in marasmic patients, particularly accompanying diphtheria, scarlatina, measles, whooping-cough, typhoid fever, etc. The cause of the infection is chiefly the diplococcus of pneumonia, in about one-half of the cases alone, in the other half associated with other micro-organisms (bacterium coli, staphylococcus pyogenes aureus, or streptococci). The process terminates either in recovery or in death, in which latter case purulent disintegration (abscess formation) or gangrene represent unfavorable complications. In rare cases the exudation is slowly absorbed or the process ends in induration (interstitial fibrous lobular pneumonia), with thickening of the septa and of the interlobular trabeculæ.

Closely related to the above-described form is inhalation pneumonia, the so-called foreign-body pneumonia. This is caused by the penetration into the bronchi of decomposable solid or liquid food, of infectious matter from the mouth, and of necrotic masses of exudation or tissue. Inhalation pneumonia likewise is found almost exclusively in the inferior and posterior segments of the lung; the initial stages resemble those of broncho-pneumonia described above; the infiltrated lobules, however, soon show a tendency to undergo gangrene and are offensive and discolored; the tissue becomes friable and dirty blackish; in place of the pus we find ichorous fluid, the covering pleura is also implicated, *i.e.*, purulent and sanious pleurisy result. Inhalation pneumonia is observed especially in patients whose power of deglutition is impaired, in grave forms of fever with clouded consciousness, in the insane, in diseases of the brain and its meninges, and largely also in patients fed artificially; it occurs, besides, when a carcinoma of the œsophagus perforates into the trachea or bronchi.

Croupous or Fibrinous Pneumonia.

(Plates 26 and 27.)

This is an acute infectious primary inflammation of the pulmonary parenchyma, caused in the majority of cases by the diplococcus pneumoniæ (Fränkel-Weichselbaum). The exudation is cellulo-fibrinous and hemorrhagic; the disease is lobar, rarely lobular, sometimes involving the whole lung, and associated with pleurisy (pleuro-pneumonia) and croupous ascending bronchitis.

Analogous to the clinical picture, the process shows anatomically a typical course. In the beginning (first stage) the pulmonary tissue appears rich in blood, juicy, swollen (engouement, stage of engorgement), with reduction of the contained air. After a short time (one to two days) red hepatization follows (second stage, see Plate 26); the doughy and spleen-like consistence changes to that of the liver; the affected lobes are voluminous, heavy, airless, and brownish-red in color; a rigid, cellulo-fibrinous, and hemorrhagic exudate being effused into the air spaces of the lung. The pleura is tense, the parenchyma rather soggy, the cut surface finely granular, since the alveolar plugs of fibrin project beyond the level of the cut surface, and on being scraped with the knife are visible to the naked eye as numerous particles like grains of sand. In serious and fatal cases a third stage follows about the seventh or eighth day, *i.e.*, gray hepatization (Plate 27); the red and brown color gives place to a gray tint, evidently owing to the rapid disintegration of the red blood corpuscles and absorption of the coloring matter. The tissue is still more juicy and friable; a creamy, almost pyoid fluid in large amounts can be scraped from the cut surface. In some cases there is a true purulent infiltration of the affected portions of the lung; rarely abscess formation and gangrene follow. The fatal issue, which occurs in about one-fifth of all cases and almost without exception in the stage of beginning gray hepatization (from the sixth to the eighth day), is usually due to oligæmia and heart failure. When recovery results, resolution and absorption of the exudate begin on the seventh day. In that case normal circulatory relations set in with active regeneration of the alveolar epithelia; the coagulated masses of exudate becoming peptonized and undergoing fatty degeneration before absorption. The exudate is gradually replaced with air and at the end of the third week the lung again appears capable of function. Only the total or partial adhesion of the layers of the pleura (adhesive pleurisy), which results from the acute exudative, and generally sero-fibrinous pleurisy, forms a permanent residue of the pleuro-pneumonia from which the patient has recovered. Many forms of pneumonia present from the start a pernicious, more septic character when streptococci and staphylococci are active in the production of the disease, or the cases become malignant in their course by secondary mixed infection. In such instances we find not rarely at the autopsy as incidental complications purulent pleurisy, endocarditis and pericarditis, meningitis, and icterus (bilious pneumonia). A rare termination is in induration and carnification, *i.e.*, the inflammatory infiltrated stroma changes into a tissue at first cellular and later fibrous; the pulmonary alveoli appear filled with vascularized, bud-like vegetations which spring from the alveolar framework and take the place of the fibrinous masses of exudate by way of substitution.

Besides idiopathic croupous pneumonia we frequently observe secondary and accidental croupous inflammation of the lung, in which numerous adhesions can be recognized, especially in debilitated, anæmic, and aged persons; instead of the firm rigid hepatization only splenization develops, by reason of a defective fibrinous exudation.

Desquamative Pneumonia.

This form is characterized by the fact that the exudate consists exclusively of proliferated and desquamated alveolar epithelia, and is largely classed with catarrhal pneumonia. The affected portions, usually the whole of the lungs or at least entire lobes, are enlarged, increased in weight, spleen-like in consistence, and rather grayish-red in color. The exudate escaping from the cut surface is tenacious, gelatinous, sometimes resembling frogs' spawn; the contained blood is usually moderate or small in amount; the pleura is not implicated. The disease rarely occurs primarily, more often secondarily, especially in connection with chronic œdema, with congestive conditions (desquamative congestive pneumonia), with acute miliary tuberculosis, with tuberculous chronic inflammation of the lung, now and then with infectious diseases (typhoid fever, pyæmia). This little-known type of pulmonary inflammation occurs in an absolutely pure form in verminous pneumonia of the domestic animals, when the pulmonary parenchyma is invaded by large numbers of the embryos of the strongyli, whose appearance resembles that of the trichinæ.

Chronic Interstitial Pneumonia.

Pulmonary Cirrhosis.

This is a chronic productive inflammation, usually observed as the termination of various inflammatory processes. It largely constitutes a kind of incomplete recovery, since there is developed from the stroma of inflamed portions of lung a fibrous proliferation, a callous and cicatricial metamorphosis. The affected parts appear more or less shrunken, of a firm consistence, they creak under the knife, are airless, anæmic, and discolored to a gray or slaty black tint (slaty cirrhosis) according to the quantity of the deposited pigment (soot). In rare cases, especially when the process has developed from croupous pneumonia, the color of the firm, connective-tissue products is rather whitish, reddish-white, or almost fleshy (induration, carnification); the newly formed connective tissue is then intra-alveolar, starting from the framework of the alveolar wall. Associated processes frequently found are bronchitis in all gradations, bronchiectases, and membranous pleurisy. In some cases we can recognize even with the naked eye that the connective-tissue deposits spring chiefly from the interlobular connective tissue, from the connective-tissue sheaths of the vessels and bronchi (fibrous perivasculitis and peribronchitis). In chronic inhalation of dust (chalicosis and siderosis) extensive cirrhotic processes also develop often in the lungs. When cavities are associated with the usual slaty cirrhosis the process is as a rule of tuberculous origin.

TUBERCULOSIS OF THE LUNGS.

(Plate 28, Figs. *a* and *b;* Plates 29 and 30.)

Pulmonary tuberculosis occurs in three main forms: 1. As acute or subacute disseminated miliary tuberculosis. 2. As tuberculous, usually recurrent fibrous or cheesy inflammation of months' or years' duration. 3. As a mixed form, a miliary eruption of tubercles being superadded to the tuberculous inflammation.

Among the organs showing a predilection for taking up and fixing the tuberculous virus from without, the lungs occupy the front rank; the tubercle bacilli enter the pulmonary alveoli with the inspired air in the form of dust. The predisposition of the lung tissue is shown by the fact that the large majority of cases of human tuberculosis begins in the lung and almost as a rule at the apex, the locus minoris resistentiæ. This peculiarity of the apex is most clearly evidenced by the numerous cases (about one-fourth of all adult persons who have not directly succumbed to tuberculosis) of healed or healing apical tubercles (Plate 28, Fig. *a*) in subjects of different ages, dead of other processes. As in persons who breathe air charged with bacilli all parts of the lungs—as in the inhalation of soot—take up about the same quantity of virus, and, as pulmonary tuberculosis begins almost without exception at the apex, we are justified in concluding that by far the greatest majority of the germs of tuberculosis which reach the lung is destroyed by the physiological forces of the organism and perishes either in the pulmonary parenchyma or within the lymph channels. Of the many thousands of tubercle bacilli which reach the lungs in the form of dust, therefore, only a few that find at the apex the conditions favoring their deposition and multiplication produce a local infection.

This predisposition of the apex of the lung is due to several factors, namely, defective function in habitual superficial respiration and stooping attitude, weakness of the respiratory muscles, epistatic and marasmic anæmia in cardiac weakness and oligæmia. The local predisposition of the apical portions of the superior lobes is also favored by lesions of the pulmonary tissue such as are produced by the continual inhalation of certain kinds of dust, especially the mineral and metallic. Finally certain constitutional, debilitating influences—anæmia, diabetes, chronic congestive disturbances, cancerous cachexia, psychical depression, imprisonment, and similar factors—have a predisposing effect or favor the rapid further spread of older latent foci (Plate 28, Fig. *a*).

Secondary tuberculosis of the lungs is found much more rarely, now and then in children when the infectious process extends toward the root of the lungs from the primarily affected peribronchial or mediastinal lymphatic glands, as it were in an inverse direction; or the secondary pulmonary tuberculosis is of hæmatogenous and metastatic origin, occurring as a local phenomenon of a general miliary tuberculosis which has arisen by way of auto-infection from any older latent focus and in which the tuberculous virus has invaded the body in any manner, usually cryptogenic.

Acute and Subacute Miliary Tuberculosis of the Lung.

(Plate 28, Fig. *b*.)

Acute miliary tuberculosis of the lung represents the typical picture of infectious bacillary tuberculosis and is characterized by the progressive new formation of multiple, miliary, infectious granulomata which occur in the stroma of the tissue. There is no mixed infection, and of the accessory processes which play so important a part in the more slowly progressive, inflammatory forms of tuberculosis we encounter only acute diffuse desquamative pneumonia and bronchitis.

The lungs appear enlarged, heavy, dark blue or brownish-red, and the pleura is transparent; through the latter we observe numerous small, sometimes barely visible gray nodules, whose centres show a slightly yellowish turbidity. The same nodules (miliary tubercles) are found abundantly scattered through the pulmonary parenchyma, which is rich in blood and succulent and whose contained air is more or less diminished. Frequently the nodules are somewhat larger in the upper than in the lower lobes, so that the conclusion appears justified that also in acute hæmatogenous auto-infection the upper lobes present more favorable conditions for the development of miliary tubercles than the better vascularized lower lobes.

Besides the acute and peracute miliary tuberculosis (Plate 28, Fig. *b*), which may terminate fatally in ten to fourteen to twenty-one days, in which the specific infectious granulomata are in the form of minute, punctiform, gray, and sometimes barely visible nodules, and the cut surface of the lung appears as if sprinkled with extremely fine sand, we occasionally observe also a subacute miliary tuberculosis of the lungs, in which the granules, ranging from the size of a pin's head to that of a hempseed, with greater yellowish turbidity in the centre, are uniformly scattered through the parenchyma.

In acute miliary tuberculosis we find as a rule somewhere in the body, most frequently at the apex of the lungs or in the lymphatic glands, an older cheesy and localized tuberculosis as the starting-point of the terminal and invariably fatal auto-infection.

Acute miliary tuberculosis of the lung, as a local phenomenon of general miliary tuberculosis with the simultaneous eruption of the nodules in numerous organs of the body (liver, spleen, and kidneys), constitutes about eight to ten per cent of all fatal cases of tuberculosis.

Inflammatory Pulmonary Tuberculosis.

There is hardly a disease which anatomically and clinically presents such multiplicity and variability in its course as pulmonary tuberculosis. Aside from acute forms which terminate fatally in a few weeks, we observe others which spread slowly in the course of months and years, relapse frequently, come to a standstill, and pass with or without mixed infection.

The reasons are largely obscure why in one case tuberculosis runs a rapid course, and in other cases advances chronically and slowly, extending through years and even decades; aside from individual resistance and disposition, accidents (erosion of vessels) and weakening influences obviously play a prominent part. A special influence unquestionably belongs to the age of the patient: in children and young persons tuberculosis has a decided tendency to run a rapid course, in older and senile patients we often observe a certain benignancy of the process, a slower progress, a diminished destructive tendency, and a greater disposition to fibrous and productive inflammations.

While a varying virulence of the germs of infection has thus far not been exactly proven, an important rôle is played by hereditary tendency, age, external noxæ (quality of the air, debilitating influences, etc.), and social conditions (occupation, nutrition, alcoholism) in the course of tuberculosis. Tuberculous phthisis, so called, is ordinarily a process complicated by mixed infection (accidental infection with staphylococci, diplococci, and streptococci); the so-called hectic fever in phthisis is usually due to an infection with streptococci and belongs to the group of septic fevers.

Pulmonary tuberculosis begins in the apex of the lung, where it causes at first a circumscribed inflammation terminating in caseation or fibrous, callous induration (slaty cicatricial foci of an irregular form, usually situated immediately under the pleura, and associated with adhesive pleurisy). The slaty and indurated foci generally contain cheesy patches from a hempseed to a cherry in size (Plate 28, Fig. *a*), which when well encapsulated frequently show a tendency to a mortar-like inspissation and calcification. In unfavorable cases we see at the margins of the cheesy patches minute gray and grayish-yellow nodules (miliary tubercles), the result of regional infection, which, growing slowly or rapidly, coalesce with the primary focus and lead to its enlargement.

From the apex of the lung the tuberculous process spreads downward first to the superior lobe, partly by continuity, partly by way of the lymph channels, or by communicating with a bronchus, by way of aspiration and intrabronchially, into previously normal portions of the lung. As a result of the intrabronchial transportation of the tuberculous virus, there occurs, in a centrifugal direction, a series of processes which must be interpreted partly as broncho-pneumonic, partly as peribronchitic tuberculous inflammations (Plate 30). In favorable cases and with a certain resistance of the patients, pre-eminently fibrous forms of peribronchitis (peribronchitis fibrosa [illegible] [illegible] [illegible] which [illegible] the healthy lung [illegible] [illegible] lobular arrangement; we [illegible] [illegible] [illegible] [illegible] [illegible] are [illegible] [illegible] grey and grayish-red [illegible] nodules [illegible] from bronchial and contiguous infection. [illegible] we find in the middle and inferior [illegible] of the lungs markedly lobular, reddish-[illegible] nodule [illegible] yellowish cheesy, usually broncho-pneumonic masses, which in the worst cases are so numerous that they become confluent and the cheesy [illegible] pneumonia changes into a lobar subacute pneumonia (Plate 35). In the latter case the lung is [illegible] heavy, and liver-like in consistence; the tissue is airless and friable, the cut surface [illegible] [illegible] and bloodless.

[illegible] pneumonia—in the form of a malignant [illegible] progressive inflammation which destroys the [illegible] tissue—therefore occurs in a lobar, [illegible] and [illegible] type; the latter represents a [illegible] [illegible] pneumonia in which the alveolar structure can still be recognized with the microscope [illegible] obliterated in true miliary and solitary tubercles (tuberculous granulomata).

The [illegible] processes which play a prominent part in tuberculosis of early course extending over months and years are the result of a chronic interstitial productive inflammation, and the tissue is usually heavily loaded with pigment (soot)—slaty [illegible] induration. The tissue at the same time is largely firm and creaks under the knife; along with it are found streaky and branching whitish connective-tissue trabeculæ which correspond to the thickened sheaths of the small bronchioles and vessels. The cirrhosis has a tendency to spread progressively from the apex to the remaining portions of the upper lobe; now and then we find the same process on a smaller scale at the tip of the lower lobe, when a descending tendency in its spread is likewise noticeable. Any possibly remaining parts of the pulmonary parenchyma present desquamative and degenerative changes about the alveolar epithelia or transitions to necrosis and caseation; such parts appear actually spotted, sometimes looking like granite. Owing to the contraction of the pulmonary parenchyma there are secondary dilatation of the bronchi and diminution of the thoracic space, especially in the upper segments.

A separate description is required to explain the formation of cavities. The first beginnings of this process can be traced to circumscribed cheesy and necrotic degenerations at the apex of the lung (Plate 28, Fig. *a*), which attack the wall of a bronchus. Owing either to the necrosis of the wall of the bronchus or to the puriform central softening of the focus, there arises an ulcerative defect in the bronchial wall, which enlarges slowly with the continual evacuation of the softened cheesy and necrotic parts. The cavity, originally the size of a hempseed or pea, increases by the advancing necrosis of the parietal layers, which is due partly to the effect of the tubercle bacilli, partly to that of accessory pyogenic germs. Should cirrhotic tissue surround the recently formed cavity, the necrotic destructive process will meet with great resistance. Rapid enlargement is often favored by the confluence of neighboring cavities; in this manner are produced larger, irregular defects with jagged walls, trabecular protrusions, and numerous excavations. While cavities in their initial stages can probably be obliterated by way of cicatricial contraction, those of the size of a pea and above possess all the conditions for a progressive increase.

In the living patient, cavities inaccessible to physical diagnosis by reason of their location or smallness, may possibly be recognized by the bacilli contained in the sputum. The formation of multiple small cavities (Plate 30) in the later and terminal stages of tuberculosis, usually associated with multiple cheesy lobular pneumonia, is markedly favored by accidental mixed infection with pyogenic organisms, so that these rapidly developing and often purulent cavities belong at least in part among the pulmonary abscesses. They are frequently found in considerable numbers scattered through diseased tuberculous lung tissue, and when subpleural in their location they cause, owing to the rapidity of their formation, not rarely a terminal pyo-pneumothorax. When multiple abscesses form, which may also result from the pyogenic effect of the toxins produced by the tubercle bacilli, the pulmonary tissue on the cut surface appears perforated like a sponge (Plate 30), whose pores and interstices are filled with pus and products of disintegration.

In all these inflammatory, fibrous, and cheesy processes we usually find scattered between and alongside the diseased portions of the lung acute and subacute miliary tubercles, in like manner as upon the covering pleura miliary tubercles and secondary tuberculous inflammations are very frequently met with.

Of course the above-described cheesy and necrotic processes not rarely involve also the vessel walls; as soon as the internal layers of the intima are attacked a plentiful invasion of tubercle bacilli occurs into the blood current, and a hæmatogenous general infection, an acute terminal general miliary tuberculosis, results. Besides, there is frequently a more benign and slower development of metastases (chronic or subchronic miliary tuberculosis), in which only isolated distant organs (particularly the spleen, kidneys, brain, bones, and joints) are implicated and at the autopsy are found to contain single or multiple larger tuberculous foci (solitary tubercles); the products of this secondary metastatic tuberculosis are very inconstant with reference to localization and duration.

In conclusion let us attempt to sketch the appearances to be observed on the post-mortem table in the majority of cases of fatal pulmonary tuberculosis. Thus in the first place we usually see on examining the pleura all possible degrees and stages of pleurisy —from the sero-fibrinous form of a few days' standing to a purulent and hemorrhagic inflammation with or without eruption of tubercles in the layers of the pleura; or else over the upper portions of the affected pulmonary parenchyma we find all grades of adhesive pleurisy with delicate spiderweb-like synechiæ or firm membranes which may reach the thickness of a finger and can hardly be detached; the lungs are usually increased in weight, their upper portions sunk in, the lower voluminous.

The parenchyma presents almost invariably the appearance of recurrent disease. At the apex and about the entire upper lobe slaty cirrhotic and indurated processes with cavities are present; with extreme frequency the apex is found changed into a cavity ranging in size from that of a hen's egg to that of a fist, which is usually torn into by the force required to remove the organ.

Aside from the large apical cavity the upper lobe often shows a larger number of irregular, older or more recent cavities; the cut surface then presents a sponge-like appearance with numerous perforations; the scanty remnants of the pulmonary tissue are more or less airless, partly showing a slaty discoloration and fibrous condensation, or they are sprinkled with recent and older cheesy foci. The middle portions of the lung contain besides patches of splenization with a gelatinous lustre and gray color (frog's spawn-like infiltration, desquamative pneumonia), or lobular cheesy friable foci, partly of a firm consistence, partly undergoing softening. The lower lobe usually contains acute and subacute sequelæ, frequently in the form of tuberculous inhalation pneumonia as lobular cheesy infiltrations or peribronchitic patches arranged in groups (agglomerated tuberculosis), along with older slaty-gray foci of a firm consistence, surrounded by a circle of acute gray miliary tubercles. The tips of the lower lobes often contain small cavities. In the intervening, still aërated tissue are isolated disseminated tubercles ranging in size from that of a millet seed to that of a pea, which are frequently visible through the pleura. Numerous transitional states between proliferative processes (miliary tubercles, infectious granulomata) and inflammatory conditions are found in many variations and combinations.

Very often there are developed in the formerly intact portions of the lung terminal accidental inflammatory processes—fibrinous, lobular, and lobar pneumonia with consecutive exudative pleurisy, unless the pleural cavity has been previously obliterated. All the numerous and variously combined alterations with regard to their localization are besides characterized by the fact that the upper segments of the lung contain chiefly the older, ulcerative, and indurative processes; the middle and lower segments, the rapidly developed terminal inflammatory processes (desquamative and cheesy pneumonia, inhalation pneumonia, acute and subacute tubercles). In some cases the destruction of the lung has advanced so far that only about one-fifth to one-sixth of the entire organ is still capable of function.

In harmony with the polymorphism of the anatomical picture and the course, which is often slow, extending over many months and years, we find in pulmonary tuberculosis, as in no other pathological process, side by side nearly all morbid conditions belonging in general pathology. I may mention only the various inflammatory processes with their numerous products and terminations, the proliferative and neoplastic processes, the retrogressive metamorphoses (fatty and hyaline degeneration, caseation, calcification); all these numerous processes occur together and successively (a true microcosmus) in recurrent pulmonary tuberculosis. This circumstance explains the difficulty of an accurate and exhaustive description of these manifold processes, which, moreover, present etiologically, with reference to the local and general predisposition, to the specific and mixed infection and intoxication, a variability and multiplicity such as occurs in hardly any other organic disease.

Syphilis of the Lung.

In the adult syphilitic processes in the lung are very rare; now and then we observe gummata, which may reach the size of a hen's egg and have a tendency to necrosis, and the formation of cavities, especially in the middle and lower portions of the lung, sometimes associated with circumscribed interstitial pneumonia. In congenital syphilis of the new-born specific processes are more frequent in the lung. First among these are gummata, which may soften and cause cavities resembling abscesses; next in order is white pneumonia, so called, in dead-born children or such as have lived but a short time after birth. In the latter the lung appears atelectatic or contains only traces of air, is of a whitish-gray or reddish-white color, with a histological structure resembling that of desquamative pneumonia. Finally, a syphilitic interstitial pneumonia occurs in children of feeble vitality; the lungs appear enlarged, of a grayish-red color, firm, with the contained air greatly diminished by reason of the smallness of the alveoli and of the cellular and connective-tissue thickening of the stroma.

Neoplasms of the Lung.

Primary new formations are very rare; now and then we observe primary carcinoma of the lung or bronchi, also enchondromata, multiple osteomata, and dermoid cysts. On the other hand the lung is frequently the seat of metastatic carcinoma (Plate 32) or sarcoma, both of them in the form of multiple, usually subpleural nodules of different sizes.

PARASITES OF THF LUNG.

Aside from vegetable microparasites which represent the regular cause of the frequent and manifold inflammations of the lung, where they evidently find few obstacles to their nidation and multiplication, animal parasites are very rare in the lung, the echinococcus being perhaps the only one.

DISEASES OF THE PLEURA.

(Plates 31 and 32.)

Injuries of the pleura usually cause the effusion of blood into the pleural sac (hæmothorax); in penetrating wounds of the chest or with simultaneous injury of the lung air frequently enters the pleural space at the same time (hæmo-pneumothorax).

Pneumothorax, the entrance of air into the pleural sac, occurs as a rule in consequence of perforation of the pulmonary pleura by cavities, abscesses, or gangrenous foci of the lung. By far the most frequent cause (four-fifths of all cases) is some ulcerative process of rapid development in recurrent pulmonary tuberculosis, usually combined with purulent pleurisy (pyo-pneumothorax). On opening the abdominal cavity the diaphragm of the affected side is seen to bulge downward; when one of the intercostal spaces is perforated the air escapes with a hissing noise. The lung is frequently correspondingly collapsed and retracted, whereby the perforation is often diminished. When older partial pleural adhesions are present at the same time, the pyo-pneumothorax is sacculated. The effect upon the surrounding organs by the abnormal accumulation of air is otherwise similar to that of a pleuritic exudate. According to the condition of the critical perforative opening, the following forms are distinguished: 1. Closed pneumothorax, when the intrathoracic air is permanently shut off by closure of the perforation during both inspiration and expiration. 2. Open pneumothorax, when the air space communicates during both inspiration and expiration with aërated lung tissue or with the external air in perforation of the thoracic wall. 3. Valvular pneumothorax, when during inspiration air enters and cannot escape during expiration. In consequence thereof the intrapleural pressure rises until at last no more air can enter. Besides these there are transitional forms.

Hydrothorax (dropsy of the pleura) is characterized by the effusion of serous fluid into the pleural cavity, the layers of this membrane being normal, smooth, glossy, and translucent. The serous transudation is usually of a wine-yellow color, bright, here and there hemorrhagic; as a rule it is bilateral and develops either as a local phenomenon of general dropsy or in connection with terminal pulmonary œdema (chronic or acute hydrothorax). Confined to one side the serous transudation is sometimes found with adhesive obliteration of the other pleural cavity, or when tumors of the mediastinum and aneurisms of the thoracic aorta compress and narrow the corresponding trunks of the pulmonary veins. According to the quantity of the transudation, which may reach two to three litres (quarts), the posterior and inferior segments of the lung appear collapsed and compressed (compression atelectasis).

INFLAMMATION OF THE PLEURA. PLEURISY OR PLEURITIS.

Pleurisy, an extremely frequent disease, occurs in numerous forms and gradations, and according to its duration is divided into acute, subacute, and chronic; or, according to its etiology, into infectious and non-infectious; or, according to its results, into exudative and productive (adhesive) forms, with numerous transitions.

Corresponding to the essentially secondary nature of pleurisy, the duration and form of the inflammation depend usually upon the variety and course of the primary pulmonary affection, especially pneumonic and tuberculous processes. In the latter we generally find recurrent forms of pleurisy, *i.e.*, over the upper parts of the lung adhesive pleurisy which has run its course; over the middle and lower portions of the lung, recent and usually exudative pleurisy. Aside from the lung (metapneumonic pleurisy) the disease develops also by contiguity from other neighboring organs—from the mediastinum, from the lymphatic glands of the root of the lung and of the mediastinum, from the pericardium, and from the abdominal cavity; now and then it may be hæmatogenous and metastatic from distant organs.

Anatomically the following principal forms may be distinguished:

1. Fibrinous or dry pleurisy. The pleura appears more or less injected, sprinkled with patches of ecchymoses, somewhat opacified, with a dull lustre and velvety aspect; a delicate, gray, fibrinous deposit may be scraped off with the knife. Very soon the sparse coagulated exudate is associated with a fluid effusion, resulting in:

2. Sero-fibrinous pleurisy, which is observed especially in fibrinous (croupous) pneumonia. Owing to the increase of the fibrinous masses of exudate, we see the layers of the pleura covered with opaque whitish or yellowish-white pseudo-membranes, 0.5 to 1 cm. in thickness, whose surface shows numerous reticulated and villous prominences. The deeper portions of the exudate which are situated directly upon the pleura rapidly present intimate adhesions to the inflamed serosa, beginning organization, and consequently a transition to productive inflammation. In favorable cases, particularly when the masses of exudate are not large, the liquid constituents are absorbed, the adjoining surfaces become permanently glued together, with obliteration of the pleural cavity, *i.e.*, adhesive pleurisy. Not rarely the sero-fibrinous exudate is mixed with blood—hemorrhagic pleurisy—which occurs especially in tuberculous, debilitated, and cachectic patients.

3. Purulent pleurisy (empyema) develops sometimes from the preceding form or presents from the beginning an infectious character, being purulent *ab initio*. In the former case there are besides the purulent effusion more or less semisoft fibrinous deposits streaked with pus, or the sero-purulent or creamy-purulent fluid is mixed with numerous flakes of fibrin.

When of long standing the layers of the pleura are invariably in a state of fibrous thickening, opaque, and covered with a pyogenic fibrino-purulent deposit. Purulent pleurisy is observed particularly in pulmonary tuberculosis and after malignant pneumonias and abscess of the lung. Perforation into the lung and into a bronchus may follow, rarely perforation outward; recovery ensues frequently after thoracocentesis and is markedly favored by operative removal of the exudate (resection of ribs). The radical operation is followed by recovery in from fifty to eighty per cent of the cases.

A variety of purulent pleurisy is sanious pleurisy (putrid empyema); the exudate is fetid and contains without exception germs of decomposition besides the pyogenic fungi. Purulent exudates may occasionally remain present for months; the exudation becomes inspissated and changed into a cheesy mass. In that event the layers of the pleura are usually enormously thickened, forming a stiff, board-like, sometimes calcified envelope around the masses of exudate. Every exudative pleurisy causes secondary alterations of the surrounding organs, among which partial or total atelectasis and consequent functional impairment of the lung occupy the front rank. With moderate masses of exudate the inferior and posterior segments of the lung are chiefly compressed and atelectatic; with abundant exudates the lung, reduced to the diameter of a small hand, lies in the shape of a flat cake-like mass upward and inward upon the root of the lung and is entirely without air. The intercostal spaces bulge outward, the diaphragm protrudes downward, the mediastinum and the pericardium are crowded toward the opposite side. As the exudation becomes absorbed the previously atelectatic portions of the lung again expand; the longer the exudate has remained and the compression has continued, the more difficult and imperfect is the expansion of the lung. The impairment of the lung by a preceding healed pleurisy will always be less in proportion to the rapidity of the course of the inflammation and of the absorption of the exudate. In the majority of cases of adhesive pleurisy—particularly with complete obliteration of the pleural space, which is occasionally discovered accidentally at autopsies—the lung is diminished in size. More marked retraction of the lung causes shrinking of the corresponding thoracic cavity, curvature of the spine (scoliosis) toward the healthy side, and descent of the shoulder on the side of the retracting pleurisy. Long-standing pleural exudations interfere with the pulmonary circulation and when the nutritive conditions are at all favorable cause dilatation and hypertrophy of the right heart, peripheral venous congestion, and frequently also chronic bronchitis. Extension of the process to the pericardium and more rarely to the diaphragmatic peritoneum is sometimes observed.

Etiology.—Although colds, overexertion, and injuries play a certain rôle (pleurisy is three or four times more frequent in men than in women), the majority of the cases is of an infectious origin. Even in the apparently benign sero-fibrinous pleurisy pyogenic organisms can sometimes be demonstrated as causes; besides it is frequently indirectly of tuberculous origin, developing by a kind of remote effect in connection with latent or demonstrable pulmonary tuberculosis or with infectious croupous pneumonia. In purulent pleurisy we find besides the pyogenic fungi (usually streptococci) frequently pneumococci and exceptionally tubercle bacilli. In long-standing empyema the micro-organisms may be absent because they have perished; in other cases (tuberculosis) the microparasites are so sparse that they can be demonstrated only by animal experiments.

Tuberculous pleurisy (Plate 31) should be clearly differentiated from sero-fibrinous, purulent, or adhesive pleurisy which is found secondarily in all possible complications with pulmonary tuberculosis. Tuberculous pleurisy, which usually runs an acute or a subacute course, is characterized by the eruption of numerous, often barely visible miliary tubercles scattered over the opacified, more or less thickened, at times somewhat fibrous pleura; the exudate is sero-fibrinous, more often hemorrhagic or purulent (mixed infection by accidental pyogenic organisms). When of long standing with a subacute course we find yellowish cheesy nodules and foci distributed through the more markedly thickened, grayish-white pleural membrane. The view which was largely entertained, that when a pleurisy runs an unfavorable course (inspissation and caseation of the exudate with its defective absorption) tuberculosis is very liable to develop secondarily both in the pleura and in the lung, is to be modified, according to the present state of our knowledge, to the effect that the majority of cases of apparently spontaneous pleurisy are either directly of tuberculous origin or arise indirectly in connection with latent and localized pulmonary tuberculosis.

In the terminal stage of fatal pulmonary tuberculosis we find almost regularly the typical picture of a recurrent pleurisy—at the apex of the lung and over the upper lobe an old adhesive, frequently membranous pleurisy; over the middle and lower portions of the lung, recent exudative sero-fibrinous or purulent pleurisy in all possible gradations as regards the extent and intensity of the process.

Neoplasms of the pleura are found most frequently in the form of multiple secondary metastatic cancerous nodules (Plate 32), which are often attached to the surface like drops of wax, or else in the form of sarcomatous nodules. Now and then we may observe diffuse carcinomatous infiltration (lymphangitis carcinomatosa) in a reticulated arrangement (Plate 32) or membranous diffuse proliferation with obliteration of the pleural cavities.

DISEASES OF THE MEDIASTINUM.

In the loose connective tissue of the mediastinum inflammatory processes are sometimes met with, usually resulting from extension of analogous processes in neighboring organs such as the pleuræ, the lungs, the pericardium, the sternum, the spinal column, and the œsophagus. When the connective-tissue investment of the parietal pericardium is the seat of inflammation the condition is known as external pericarditis or mediastino-pericarditis. More rarely we may discover limited inflammations in the form of abscesses, for instance, in the anterior mediastinum by extension of an inflammation from the cervical connective tissue or in caries of the sternum, in the posterior mediastinum by purulent liquefaction of lymphatic glands; such suppurations may in turn cause secondary pericarditis or pleurisy.

Of tumors, aside from secondary carcinomatous and sarcomatous diseases of the lymphatic glands, we occasionally find primary sarcomata sometimes of large size, more rarely dermoid cysts. Mediastinal sarcomata (usually fibro-sarcomata or round-celled sarcomata) extend to the neighboring organs; their effect upon the latter largely coincides with those of aneurisms of the thoracic aorta—compression of the main bronchi, of the large veins, and of the vagus nerve.

Digestive Apparatus.

DISEASES OF THE ORAL CAVITY AND OF THE TONGUE.

Hemorrhages of the oral mucous membrane are frequently observed, especially from the gums in scurvy and hæmophilia or the hemorrhagic diathesis.

Inflammatory processes (stomatitis) occur in various forms and gradations:

Desquamative or catarrhal stomatitis, with abundant proliferation and desquamation of the epithelia and increased secretion of mucus. The mucous membrane appears swollen, reddened in spots and streaks; the papillæ are more prominent than normal. The causes are mechanical, thermal, and chemical irritations, the eruption of teeth, acute infectious diseases (measles, scarlatina, small-pox), inflammations, especially of the pharynx, which have extended to the oral mucosa.

Chronic catarrhal stomatitis, usually associated with chronic pharyngeal catarrh, in drinkers and excessive smokers. There are grayish-white spots of thickening on the mucous membrane, a tough mucous secretion, and a whitish coating of the tongue, with an abundant development of fungi (leptothrix buccalis).

Stomato-mycosis (thrush), caused by a mould fungus (oidium albicans). This is a superficial mould mycosis of the epithelium, occurring mainly in infants artificially fed, more rarely in nurslings at the breast. Upon the surface of the tongue and on the palate we observe white, at first insular, later confluent membranes; the epithelial layer on the affected points is thickened and penetrated by mycelia and spores. The pseudo-membranes are at first easily detached, later with difficulty. As a rule, the fungus does not pass beyond the epithelium.

Stomatitis vesiculosa, pustulosa, et aphthosa. An inflammation of the oral cavity associated with the formation of vesicles, pustules, and ulcers, observed now and then in small-pox, varicella, and when the poison of the foot-and-mouth disease of cattle is transmitted by the ingestion of the unboiled milk of animals affected with that disease.

Purulent stomatitis in the form of circumscribed focal processes in the gums in connection with carious teeth is more frequently observed. Parulis is the term applied to abscesses, associated or not with inflammation of the dental periosteum, which are located between the gums and the maxillary bones.

Ulcerative stomatitis occurs now and then upon the gums and the adjoining portions of the mucosa of the lips, cheeks, and tongue in scurvy and mercurial poisoning, especially in debilitated patients.

In scurvy the gums present a dull gray, discolored appearance; the mucous membrane is reddened and tumid; the teeth are loosened. Owing to the disintegration of the discolored gums, ulcers with lardaceous bottom sometimes develop and bleed readily. In grave cases ulcerative processes of the lips and cheeks, secondary periostitis, and necrosis of the bones may be superadded.

Toxic stomatitis, due to mercurial poisoning (medicamentous or in certain occupations, especially the manufacture of mirrors), presents appearances similar to scurvy. Allied changes result from chronic lead and phosphorus poisoning.

Syphilis of the oral cavity may occur in the form of the primary lesion as a result of direct or indirect infection. More commonly, as the result of the general infection, we find in the oral cavity syphilitic papules or patches in the form of roundish, reddened, and slightly prominent lesions; subsequently these efflorescences present a milky opacity with an almost nacreous lustre and sometimes intense reddening. Owing to the proliferation of the papillæ and follicles, the surface is uneven. From the confluence of such papules extensive ulcers may result, at the angles of the mouth in the form of irregular fissures, so-called rhagades. The ulcerative processes are sometimes due to the breaking down of gummy formations, especially upon the tongue. Ulcers on the hard palate may be connected with syphilitic periostitis or ostitis; in that event deep-seated caries and necrosis of the bone, perforation, and communication with the nasal cavity (cleft palate) are found as complications. Smooth atrophy of the base of the tongue indicates former syphilis which has run its course.

Gangrene of the cheek (noma, water canker) develops now and then apparently spontaneously in very cachectic children suffering from grave infections (typhoid fever, scarlatina, measles, tuberculosis) and living under unfavorable hygienic conditions.

Neoplasms of the organs of the mouth are rather frequent, especially carcinoma of the lips and tongue. Cancer of the lip, most often situated on the lower lip, occurs, like cancer of the tongue, chiefly in men, rarely in women (10:1); the smoking of acrid tobacco, especially in clay pipes, and shaving with dull razors are given as the main causes of cancer of the lip. Superficial and deep forms are distinguished. Cancer of the tongue commences, as a rule, at the margins.

Ranula is the term applied to roundish or oval cysts with tenacious contents, situated under the tongue close to the frenulum; they are retention cysts due to occlusion and dilatation of the mucous glands (Blandin-Nuhn glands) located near the tip of the tongue. Or else they develop as retention cysts from Wharton's duct or from the efferent ducts of the sublingual glands, when these passages are obstructed by inflammatory processes or calculi. Besides, we find on the floor of the mouth lymphangiomata, dermoid cysts, branchial cysts (congenital cystic hygromata), which crowd upward from the neck and lift the floor of the oral cavity.

Connective-tissue tumors about the cavity of the mouth are sometimes observed; the most frequent forms are fibromata and sarcomata springing from the connective-tissue portions of the mucosa, from the submucosa, from the periosteum and bones of the maxilla (periosteal sarcomata or myelogenous sarcomata). Tumors developing on the gums are known collectively as "epulis."

DISEASES OF THE PHARYNX

Inflammatory processes are very frequent upon the pharyngeal mucosa.

Acute catarrhal pharyngitis (angina catarrhalis) develops idiopathically or secondarily, especially with scarlatina, measles, and variola. The mucous membrane is reddened, swollen, and covered with a tough mucous or muco-purulent deposit, particularly upon the soft palate and the enlarged tonsils; the follicles are usually enlarged (lacunar tonsillitis). When the purulent secretion is more abundant, the crypts of the tonsils are filled with masses of pus which exude spontaneously over the surface or are evacuated by pressure. The pathogenic fungi usually found are staphylococci or streptococci.

In chronic pharyngitis, besides the diffuse thickening of the mucous membrane, the follicles are greatly swollen (pharyngitis granulosa); the palatal tonsils are usually enlarged, and the crypts filled with yellowish puriform masses and plugs. The enlarged tonsils in chronic inflammation appear firm, discolored to a more or less slaty gray, and contain thickened, mortar-like or calcified plugs. The tonsils by reason of their structure (deep indentations of the mucous membrane between the follicles, physiological defects of the epithelial investment, with the continual emigration of white blood corpuscles, which appear in the oral secretion as so-called mucous corpuscles) are evidently very prone to take up infectious matters, and, like a sponge, to suck up pathogenic germs and thus fix objects in transit.

Diphtheroid Angina, Angina Necrotica.

This occurs sometimes in very young and debilitated children living under unfavorable hygienic conditions, and especially often in connection with scarlatina or measles, more rarely with typhoid fever, and can almost invariably be traced to streptococcus infection. The anatomical changes resemble those in true diphtheria.

The process, as a rule, springs from the tonsils or from the mucous membrane of the nasopharynx; more rarely it spreads to the larynx and trachea. The diphtheroid crusts and membranes, which are usually thin, adhere firmly to the underlying tissue.

Pharyngeal Diphtheria—Diphtheria Faucium.

This is likewise a specific, necrosing, and exudative process, caused by the Klebs-Loeffler bacillus. Together with redness and swelling of the mucosa there appear gray and whitish spots, especially on the tonsils, soft palate, and uvula, which consist of a cellulo-fibrinous infiltration of the superficial layers of the mucous membrane. The fibrinous infiltration then extends into the deeper layers of the mucous membrane and undergoes hyaline degeneration as do the blood-vessels; hyaline plugs and masses are formed. Besides the escharotic spots, in place of the necrotic epithelium croupous membranes form, which tend to spread more superficially. After the eschars are cast off, shallow ulcers remain with eroded, sometimes jagged margins and bottom.

quires a special predisposition, which is very frequent in children and rare in adults; the blood serum of persons having no predisposition possesses antitoxic properties.

Diphtheroid affections without specific bacilli do not belong to the type of true diphtheria, and wherever they occur (not only in the fauces but also on other mucous membranes, such as that of the bladder and of the intestine) should be designated as diphtheroid or simply as necrosing processes.

In true pharyngeal diphtheria the mortality may reach fifty to sixty per cent; in diphtheroid affections and other anginas at most one to four per cent.

Rare though very malignant forms of infectious angina are:

ERYSIPELATOUS AND PHLEGMONOUS PHARYNGITIS.

Both are diffuse inflammations of the mucosa and submucosa due to streptococcal infection. The mucous membrane is greatly swollen, especially at the aditus laryngis (acute inflammatory œdema of the glottis), and reddened. If the exudation is purulent the affected parts appear pale yellowish and almost fluctuating; on incision an abundant sero-purulent infiltration may be expressed. The tonsils are often likewise markedly swollen. Such cases of malignant diffuse streptococcal angina usually end fatally in a short time (sometimes one or two days); they originate either primarily, as it were cryptogenetically without demonstrable point of entry, or secondarily from purulent or sanio-purulent processes in the vicinity of the pharynx, for instance, from a deep-seated abscess at the base of the tongue, from angina Ludovici of the soft parts about the floor of the mouth or about the larynx, and from retropharyngeal, parapharyngeal, or prevertebral abscesses (Plate 34).

Syphilitic ulcerations and cicatrices are found now and then in the pharynx and at the base of the tongue. Usually as a sequel of an angina with or without specific characters we find patches, flat thickenings of the mucosa, rarely gummata, and finally shallow lardaceous ulcers, often in connection with syphilis of the nose.

Tuberculous ulcers in the pharynx are rare; most frequently they spread upward from the larynx; in general the ulcerations resemble those of the larynx.

Neoplasms of the pharynx are rare, aside from lymphomata of the lymphoid organs, and from connective-tissue polypi which develop downward from the walls of the choanæ or from the base of the skull. Quite exceptional is primary carcinoma of the pharyngeal mucous membrane; secondary cancer derived from the upper part of the œsophagus, from the larynx, or from the thyroid gland is occasionally met with.

DISEASES OF THE SALIVARY GLANDS.

Inflammation of the parotid gland (parotitis) occurs as a primary and epidemic disease (mumps). The gland is swollen, the exudate cellulo-serous; a viscid muco-purulent secretion accumulates in the efferent duct. The usual termination is in recovery.

Besides, an infectious purulent parotitis develops secondarily in connection with certain infectious diseases, such as typhoid fever, scarlatina, etc. Formerly the cause was ascribed to a hæmatogenous metastatic inflammation, but more probably the disease is due to the entrance of septic germs from the mouth through Steno's duct into the gland tissue, where they set up a purulent or sanio-purulent parotitis. The scanty ingestion of food and the diminished or arrested secretion of saliva greatly facilitate the penetration of pathogenic germs. The gland is swollen, firm and fleshy in appearance, and often infiltrated with a sero-purulent mass. The inflammation may terminate in abscess formation, partial sloughing of the gland, perforation of the pus pockets into the oral cavity, or externally, or into the external auditory meatus. This glandular suppuration or sloughing may also lead to a general embolic septicopyæmia.

Neoplasms in the parotid gland are not rare, especially sarcoma in numerous modifications, namely, fibro-, myxo-, chondro-, and adeno-sarcoma; more rarely carcinoma.

DISEASES OF THE ŒSOPHAGUS.

Acute œsophagitis, catarrhal inflammation of the gullet, is a very rare disease. The inflammatory changes, most closely resembling those of the corresponding affections of the oral cavity, are slight, being chiefly proliferation and increased desquamation of the epithelium. The causes may be mechanical (foreign bodies), chemical (strong alcoholic liquors), and thermic irritations; rarely processes extending down from the pharynx or up from the stomach. Recovery is usually rapid.

Other inflammations are uncommon; now and then a purulent inflammation of the submucosa may be due to the effect of foreign bodies. Ulcerations are also rare except perhaps in the lowest segments; in the cardiac portion peptic ulcer may result from the influence of the gastric juice. Ulceration may, however, occur also in connection with—

Toxic œsophagitis, caused by the ingestion of acids and alkalies, in all possible gradations from simple reddening to extensive eschar formation. After the moderate action of acids the mucous membrane is discolored gray or yellowish and wrinkled; after the action of the alkalies it is more brownish and soft; after intense corrosion the mucous membrane appears changed into a brown or black friable mass. Unless death ensues rapidly, the eschars are thrown off and are followed by ulceration, purulent inflammation, and the formation of cicatrices and stenoses.

Necrosing and ulcerative œsophagitis may also be due to foreign bodies and to various local processes which implicate the wall from without, *e.g.*, suppuration of mediastinal and peribronchial lymphatic glands, aneurisms of the aorta, prevertebral and retropharyngeal abscess in vertebral caries, etc.

Diverticula of the œsophagus are sac-like, circumscribed dilatations, two forms of which are distinguished:

(1) Pressure diverticula, usually situated in the upper part of the œsophagus where it merges into the lower pharynx. They result by pressure from within (during deglutition); owing to excessive stretching and partially diminished resistance of the muscular coat, the mucous membrane is forced through the

latter somewhat like a hernia and gradually dilates to a larger sac into which food continually penetrates.

(2) Traction diverticula, whose frequency is greater than that of the former, are more funnel shaped, are usually situated at the anterior wall of the œsophagus near the root of the lung, and result from traction from without, exerted by lymphatic glands which have been inflamed and gradually become sclerosed and shrunken, thus dragging on the adjoining wall of the gullet and producing dilatations and diverticula. Owing to the accumulation of food at the bottom of the latter, inflammatory processes are sometimes set up and cause perforation and sloughing in the surrounding structures (mediastinitis, pleurisy, and pulmonary gangrene).

Carcinoma of the œsophagus (Plate 35) is more frequently primary than secondary.

It begins as an insular process and has a tendency to spread as a ring or belt until it finally infiltrates the wall for a distance of 5 cm. and more. Ulcerative disintegration sets in early and is ultimately followed by perforation into the surrounding organs—trachea, bronchi, lung (inhalation pneumonia), pleura, large arterial branches, or the mediastinum. Cancer of the œsophagus is located chiefly in the middle and lower third and attacks men more often than women (5:1). The secondary stenosis may again disappear in the later stages, owing to the progressive destruction.

Cancer of the œsophagus develops now and then secondarily by contiguity from the cardia, the pharynx, or the thyroid gland.

DISEASES OF THE STOMACH.

Cadaveric softening (gastromalacia) is often found in the region of the fundus; the wall for a variable distance is changed into a friable slimy mass which tears on touch or on removal of the organ.

Circulatory Disturbances.—Hyperemia of the mucosa is found in a circumscribed form as red spots, frequently in consequence of hypostasis, particularly in the region of the fundus and on the posterior wall (Plate 36); it may be mistaken for focal hemorrhages.

Hemorrhages into the gastric mucous membrane may be often observed. The hemorrhage occurs by diapedesis in connection with congestive or inflammatory hyperæmia or with venous stasis, the mucosa showing a corresponding capillary filling with bright or dark blood. The hemorrhages may also result from emboli, when they are multiple and sharply circumscribed, usually in spots or streaks; associated with them are superficial shallow epithelial defects or losses of substance, *i.e.*, hemorrhagic erosions of [illegible]; these are met with in septic infec[illegible] [illegible]tions, as infectious emboli. Larger [illegible] the lumen of the stomach are gen[illegible] [illegible] ulcerative processes; they are de[illegible] [illegible] ulcers or from eroded larger [illegible] bottom of round gastric ulcers; [illegible] the effused blood forms a coffee-[illegible] mass, owing to the influence of [illegible].

[illegible] may also have been swal[illegible] [illegible] of the base of the skull.

Wounds of the stomach are caused by bullets or stabbing; owing to the escape of the gastric contents into the abdominal cavity a fatal perforation peritonitis follows within a few days.

INFLAMMATION OF THE STOMACH, GASTRITIS.

Acute catarrhal gastritis is due to numerous toxic, mechanical, and thermic influences. The mucous membrane is greatly injected, swollen, more folded than normal, and covered with abundant, tenacious, glairy mucus. A special form is caused by toxic agents (phosphorus, arsenic), *i.e.*, parenchymatous gastro-adenitis; the mucous membrane is not so red but of a turbid, somewhat yellowish appearance; the glandular epithelia are enlarged, with a granular opacity; in the course of a few days the cloudy swelling passes into a fatty degeneration of the glandular epithelia.

Chronic gastric catarrh (chronic gastritis) causes changes with special preference in the region of the pylorus and presents various forms, namely:

In simple chronic gastritis the mucous membrane is diffusely thickened, swollen, and corrugated, particularly near the pylorus; the submucosa is similarly thickened and proliferated.

Very often we observe in connection with chronic diseases of the liver and heart (cirrhosis of the liver, valvular defects, fatty heart, and disease of the heart muscle) a chronic congestive catarrh of the stomach. The mucosa is swollen, dark brownish-red or cyanotic in color, full of blood, succulent, and covered with abundant masses of mucus. As a rule, the same condition exists at the same time in the intestinal canal, *i.e.*, chronic congestive gastro-intestinal catarrh.

In other cases the surface of the thickened mucous membrane is covered with flat elevations and low verrucose protuberances (état mamelonné). In this hyperplastic and sclerosing form of gastritis hypertrophicans prolifera we may find, when the disease has reached an advanced stage, true warty elevations (gastritis verrucosa), or even larger polypoid proliferations (gastritis polyposa, Plate 37).

As a result of oft-repeated hyperæmic conditions with their associated hemorrhage by diapedesis we may sometimes find a diffuse or partial pigmentation of the mucous membrane, which is of a lead gray or slaty gray color, and again most markedly so in the region of the pylorus. On microscopic examination the mucous membrane is seen to be sprinkled with numerous granules of melanin — gastritis chronica pigmentosa.

As a sequel of chronic gastritis there is developed, though rarely, a connective-tissue hypertrophy of all the coats of the stomach associated with shrinking of the organ (benign sclerosis of the stomach)—a condition which is very similar to diffuse infiltrated gastric cancer. In doubtful cases the state of the lymphatic glands and the microscopic examination will settle the diagnosis. The stomach is contracted to the diameter of the intestine and the walls may reach the thickness of the little finger and are hypertrophic.

Croupous, diphtheritic, and phlegmonous inflammations of the stomach are very rare and the same remark applies to ulcerative tuberculous gastritis (Plate 38).

ULCER OF THE STOMACH.

Besides the above-mentioned hemorrhagic erosions a specific formation of ulcers occurs. Such ulcers develop without preceding infection or necrosis, and in connection with nutritive disturbances (hemorrhage, anæmia), the loss of substance being due to spontaneous digestion (peptic ulcer, Figs. 16 and 17).

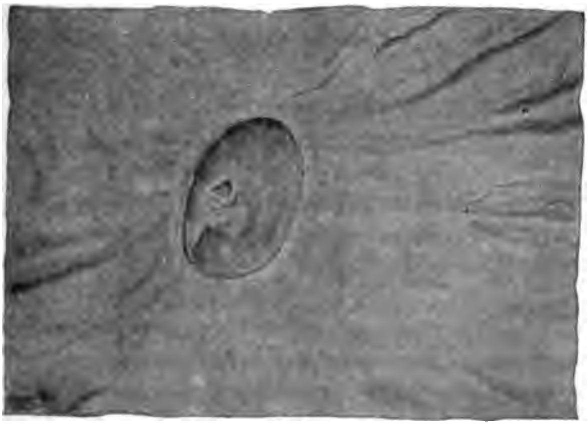

FIG. 16.—Round Ulcer of the Stomach. Ulcus rotundum pepticum. At the bottom of the ulcer, situated in the pyloric region on the posterior wall of the stomach, is an eroded vessel from which fatal hemorrhage resulted.

The principal characteristics of this aseptic, simple, or round ulcer (ulcus ventriculi simplex, rotundum) are as follows: They are located mainly on the posterior wall of the pyloric region and on the lesser curvature, more rarely at the pylorus, and only exceptionally about the cardia, the greater curvature, or the fundus. The size ranges from that of a nickel to that of a dollar. The margins are sharp, show no trace of reddening or swelling, and appear as if punched out. The defect in the mucous membrane is the largest, it is smaller in the submucosa, and least in the muscular tunic; thus the ulcer forms a funnel which narrows toward the outside. The margins resemble steps or a terrace in their configuration, though the axis of the ulcer is not always vertical but frequently passes obliquely through the wall of the stomach. As a rule, only a single ulcer is found, now and then several in various stages of development may be present; sometimes the ulcers are oblong or irregular in outline, especially at the pylorus, where they also may spread like a belt and cause stricture of the opening.

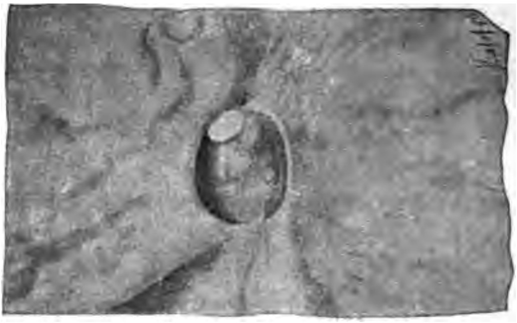

FIG. 17.—Round Perforating Ulcer of the Stomach. The ulcer with sharp margins has completely destroyed the walls of the stomach; the bottom of the ulcer is formed by adherent thickened tissue; to the left and above is a lentil-sized perforation. Death from acute purulent perforation peritonitis.

The termination varies. The smaller ulcers in particular often heal with the formation of stellate and radiating puckered cicatrices (Fig. 18). Unfavorable and almost uniformly fatal terminations are hemorrhage from small arterial twigs, barely the size of a knitting-needle, which pass at the bottom of the ulcer and are eroded laterally (Fig. 16); or perforation into the peritoneal cavity, escape of gastric contents, and fatal perforation peritonitis within a few days (primary perforation). When perforation threatens while the ulcerative process invades the muscular tunic, a reactive and adhesive circumscribed peritonitis (perigastritis) sometimes occurs on the corresponding point of the peritoneum, with resulting agglutination of the danger spot to surrounding organs, especially to the pancreas and to the retroperitoneal connective tissue. While the margins directed toward the peritoneal cavity are loosely or intimately adherent, the ulcer may penetrate into the agglutinated organs and cause considerable losses of substance, until at last, owing to an unfortunate accident

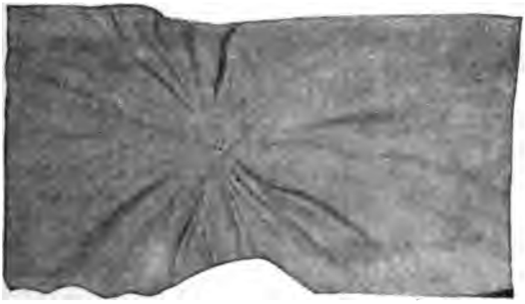

FIG. 18.—Stellate Cicatrix of the Stomach. A healed round ulcer. Starting from the whitish opaque centre, the seat of the original ulcer, we observe numerous radial processes in fine folds. At the point of the healed ulcer the wall of the stomach is somewhat thinned and faintly translucent when held up to the light.

tinuous motion. The marginal adhesions and agglutinations give way and secondary perforation ensues from some part of the margin (Fig. 17). In rare cases adhesion may take place to the anterior abdominal wall, to the liver, spleen, transverse colon, and duodenum, with formation of a [illegible] fistula when perforation occurs into an adjoining segment of the intestine.

The causes of gastric ulcer are not completely elucidated. Besides local circulatory disturbances (hemorrhages, hemorrhagic erosions, ischaemia and embolism), which favor partial self-digestion and furnish to the gastric juice, as it were, the first points of attack, some part is played by lesions of the mucous membrane of the organ (thermic and toxic influences, excessively hot food, whiskey, coarse indigestible aliment) and mechanical factors such as tight lacing.

Anomalies in the composition of the blood, chlorosis and anaemia among constitutional factors, undoubtedly favor the extension of the ulcer and prevent its healing. Badly nourished persons are more frequently attacked. In women round gastric ulcer is three times as common as in men; relapses are often observed. The cicatrices predispose to the development of carcinoma. When the ulcers and cicatrices are located at the pylorus they cause stenosis of the latter and secondary gastrectasia.

Cicatricial contraction in the middle of the stomach gives rise to hour-glass deformity, the organ being divided into two parts communicating by a narrow opening.

NEOPLASMS OF THE STOMACH

Cancer of the stomach is very frequent, constituting from one-fourth to one-third of all carcinomas; men are attacked more often than women (7 : 5). The disease is most common between the fortieth and sixtieth year of life, there being hardly any difference between the various classes of society. The sites of predilection of gastric cancer are the pylorus and its neighborhood and the lesser curvature; the cardia, the anterior and posterior wall are more rarely affected. As a rule, the tumor is circumscribed, exceptionally it is spread diffusely over the greater portion of the gastric wall (infiltrated cancer).

The typical forms are medullary or glandular cancer, scirrhus or hard cancer, and colloid cancer.

At first the carcinoma is always parietal and tends to spread superficially and, particularly at the pylorus, annularly.

Medullary carcinoma usually presents tumid, elevated margins, and is semi-soft in consistence, with an ulcerating surface of irregular form in the centre. In some cases the cancer constitutes a fungoid mass of a reddish color, is not very prone to break down, and is quite vascular (carcinoma telangiectodes), with a tendency to hemorrhages. The gastric contents resemble coffee grounds and consist of altered blood.

Hard cancer (scirrhus or fibro-carcinoma) may be circumscribed to the pylorus; the infiltrated wall creaks under the knife, and hardly any juice can be scraped from the thickened wall. Ulceration is often very limited; shallow losses of substance may be seen on the mucous surface of the affected portion. In other cases this form is more diffuse and spreads over the greater part of the stomach, which is usually contracted (cirrhosis)—a condition sometimes closely resembling simple, inflammatory, non-cancerous, or benign cirrhosis.

Colloid cancer is more apt to form diffuse infiltration; the reticular and alveolar stroma incloses numerous lacunæ containing transparent gelatinous drops; on the cut surface the firmly infiltrated and thickened wall of the stomach sometimes presents a honeycomb appearance. Colloid cancer tends to extend by continuity to neighboring organs (the greater and lesser omentum) and to the lymphatic glands (Plate 39).

In very rare cases only do we find the cancer confined to the stomach and the lymphatic glands not involved. The condition of the latter (especially the epigastric glands) by the way, and, as a rule, both macroscopically and microscopically, greatly facilitates the differential diagnosis between cancer and other ulcerative processes. All the above-named forms of carcinoma tend to pass through the walls of the stomach and to involve the peritoneum; they also have in common the tendency to cause stenosis of the pylorus, gastrectasia when seated at the latter point, recurring minor hemorrhages, and perforation of the wall of the stomach. The last-mentioned accident is retarded and postponed by adhesion and agglutination of the weakened portion to the adjoining structures (pancreas, liver, duodenum, transverse colon, more rarely the anterior abdominal wall). Perforation into the colon with the formation of a bimucous gastro-colonic fistula is uncommon, and still rarer is adhesion to the anterior abdominal wall and perforation outward, *i.e.*, an external gastro-abdominal fistula. The fatal issue is usually due to general exhaustion and inanition.

Numerous etiological factors have been cited as favoring the development of gastric cancer: preceding ulcers and cicatrices; thermic, chemical, and mechanical, oft-recurring lesions of the mucosa. As upon other mucous membranes (oral cavity, colon, female genitals) gastric cancer develops by preference at points where chronic inflammatory processes and ulcerations are most frequent, *i.e.*, at the pylorus and in its neighborhood and at the lesser curvature, so that some connection between such irritative conditions and the development of carcinoma is not to be denied.

Other neoplasms (sarcoma, myoma, lipoma) are extremely rare in the stomach.

Foreign Bodies.—Bones, needles, and coins, which sometimes reach the digestive canal by accident or design, often pass safely through the pylorus and the intestine, or they may remain a long time in the stomach, or they may pierce the wall, causing fistulæ and perforation toward the surface.

DILATATION OF THE STOMACH, GASTRECTASIA.

This condition occurs as acute dilatation in consequence of overloading of the stomach or of serious dietetic errors (dilatatio ex ingestis), of excessive development of gas, and more rarely of affections of the muscular tunic (central paralysis?). Oft-repeated acute dilatations and muscular insufficiency may result in chronic permanent dilatation.

More frequently we meet with chronic dilatation of the stomach, which should be clearly differentiated from physiological megalogastria in healthy persons who habitually consume large quantities of food and drink. The condition may develop primarily, as an atonic muscular gastrectasia, in connection with exhaustion in typhoid fever, chlorosis, and neurasthenia, or with chronic catarrhal gastritis; owing to the diminished tone of the muscular coat the stomach relaxes and dilates. Quite frequently we find secondary or mechanical gastrectasia which develops from stenosis of the pylorus caused by cancer, cicatricial contraction, and hypertrophy of the muscular tunic.

Contraction and shrinking of the stomach develop in stenosis of the cardia, in prolonged starvation, in cirrhotic and hypertrophic thickening of the gastric wall, in diffuse infiltrated carcinosis of the walls, or in diffuse neoplasms of the peritoneum; circumscribed contraction and hourglass constriction, in cicatricial contraction of the middle portion after the healing of a round ulcer.

DISEASES OF THE INTESTINE.

As the functional disturbances of the intestine go hand-in-hand with the corresponding functions of the stomach, so we also find on pathologico-anatomical grounds a certain coincidence. The noxæ caused by faulty food and drink are the same, as are often also infectious and toxic influences; the results of circulatory disturbances due to the liver or the heart are similar in the stomach and the intestinal canal. Primary, especially malignant tumors occur in the intestine as they do in the stomach, but more rarely. A group of infectious diseases, such as typhoid fever, cholera, and dysentery, is located primarily in the intestinal mucosa; tuberculosis likewise is very frequent, though as a rule only secondarily.

Among the circulatory disturbances the most frequent form is congestive hyperæmia which occurs with diseases of the liver, thrombosis of the portal vein, and affections of the heart. The mucous membrane is dark bluish-red, swollen, very succulent, and friable; the contents are often mixed with blood; abundant gray, glassy masses of mucus cover the mucosa (chronic congestive catarrh). In embolic or thrombotic occlusion of the mesenteric arterial twigs we note the development, often over large segments of the intestine, of a hemorrhagic infarction with effusion of blood into the lumen of the affected portion. Pronounced acute congestive hyperæmia in circumscribed segments of the intestine occurs regularly in incarceration, owing to the occlusion by compression of the efferent mesenteric veins; this is often very rapidly followed by a sero-sanguinolent transudation into the hernial sac and into the lumen of the intestine.

Inflammation of the Intestine, Intestinal Catarrah, Enteritis.

This occurs in so many forms and gradations that a comprehensive description is hardly possible.

Catarrhal enteritis or intestinal catarrh varies from a slight catarrh to a true inflammation and is either confined to some segments of the bowel or occurs diffusely in the small and large intestine. Reddening, swelling, increased accumulation of mucus, augmentation and fluidity of the contents are the most important characteristics.

A particularly dangerous form is infectious gastroenteritis in infants (cholera infantum, summer diarrhœa of children). This is due to the effect of saprophytes which by their fermentative activity produce poisonous chemical substances (ptomains), causing grave and frequently fatal auto-intoxication. It is probable that a continuance of ectogenous processes of decomposition in bottle-fed children plays a part in the affection. While in the upper portions of the bowel abnormal lactic-acid fermentation and decomposition of sugar predominate, in the lower portions alkaline fermentation and decomposition of albuminous bodies take place. At the autopsy we find the intestinal contents thin-fluid and usually of a penetrating offensive odor; the mucosa is pale, the epithelium loosened and in many places desquamated, the follicles are largely swollen, *i.e.*, a septic and septiform enteritis. While the gastric juice by reason of its contained hydrochloric acid has some bactericide and antiseptic properties, these are only limited, and the digestive ferments exert no protective influence in this direction. Under the influence of the disturbed chemical activity, of the abnormal processes of decomposition and fermentation, harmless intestinal micro-organisms become pathogenic (facultative parasites); the serous transudation and the increased intestinal secretion contain much serum-albumin and mucin, greater quantities of oxygen, and hence favorable conditions for the abnormal augmentation of the bacteria.

Besides this intestinal infection, in which food, drink, and perhaps the saliva furnish the vehicles, there is also a hæmatogenous infection of the intestinal mucosa; in this manner, for instance, a secondary metastatic septic enteritis develops in general sepsis.

In chronic intestinal catarrh (Plate 40) the mucous membrane is usually thickened and of a slaty gray color, especially in the chronic congestive catarrh that develops secondarily in chronic affections of the liver and heart. In some cases the mucous membrane is thickened together with the remaining parietal layers, and polypi develop, *i.e.*, proliferating hyperplastic enteritis, enteritis polyposa. Or else, following the acute enteritis of children, with general cachexia and anæmia, we find an atrophy of the glandular portions of the mucosa, *i.e.*, chronic enteritis atrophicans.

Follicular enteritis or follicular catarrh is the term applied to an intestinal inflammation in which the follicles are largely involved, especially in the lower ileum and the first portions of the colon (Plate 41). The mucous membrane shows the ordinary inflammatory changes and, owing to the swelling of the follicles, a grater-like appearance. In some severe cases the follicles suppurate and break down, forming small abscesses (follicular abscesses) which perforate into the lumen of the intestine, and multiple follicular ulcers develop. These ulcers are small, from a lentil to a hempseed in size, and may coalesce. When such lesions are localized in the colon the cases are sometimes designated as follicular dysentery.

Croupous enteritis is very rare.

Diphtheroid and necrosing enteritis (Plate 42) is characterized by marked swelling, injection, reddening, serous and sero-cellular infiltration of the intestinal wall, especially the mucosa and submucosa; the mucous membrane is enormously swollen, tumid, almost fluctuating, and divided into transverse, flabby ridges. The upper layers of the mucous membrane may be in a condition of commencing or advanced eschar formation; at first appearing as if sprinkled with bran, later covered with dirty gray and dull yellowish eschars. Such severe and usually fatal forms of diphtheroid enteritis (colitis and proctitis) are met with in true dysentery and also sometimes in acute medicinal mercurial poisoning, in which cases the poison gains entrance to the body through the skin, the subcutaneous tissue, the serous and mucous membranes, or through internal administration, and is excreted by the intestine, especially the colon (Plate 43). In uræmia likewise, by reason of the auto-intoxication and the excretion of urea into the lumen of the bowel, we observe secondary toxic enteritis (enteritis uræmica), which may sometimes go as far as eschar formation in the upper layers of the mucosa; finally, a diphtheroid enteritis (colitis and proctitis) occurs in sepsis (Plate 44).

Circumscribed necrosing and ulcerative inflammations of the intestinal wall develop sometimes in connection with stagnation of the contents of the bowel, especially in the colon; such stagnation, acting mechanically, infectiously, and toxically, producing fæcal or stercoral pressure necrosis, which is particularly frequent and malignant in the vermiform appendix, where fæcal concretions (coproliths) set up necrosis of the mucosa and the rest of the intestinal wall and finally cause perforative appendicitis (Plate 47, Figs. *a* and *b*). Similar processes, though they terminate more rarely in perforation, are occasionally found in the cæcum (typhlitis and perityphlitis). As regards the sequelæ of ulcerative appendicitis the following are evidently the determining elements: the situation and attachment of the vermiform appendix, the nature of the necrosing factor (mechanical and benign, or infectious and malignant) and hence the rapidity of the perforation; in other words, the more rapid the destruction and perforation of the wall of the appendix the less time is given for adhesion and encapsulation of the local process and the more quickly will dangerous diffuse peritonitis develop.

After inflammatory processes of various kinds we find rather frequently in adult cadavers a partial or complete obliteration of the vermiform apendix. Now and then we may discover, as at other points of the bowel, purulent and sanious periappendicitis (perityphlitis) without perforation of the appendix, in which cases septic germs emigrate through the diseased intestinal wall.

Finally, chronic inflammations of the intestine occur sometimes about the rectum and assume very different forms. Ulcerative processes are met with along with hypertrophy of the mucous membrane (proliferating and stenosing proctitis), thickening of the muscular tunic and the adjoining soft parts, which are often undermined with fistulæ, especially toward the anus and perineum; at the same time we sometimes find fistulous passages, in women the formation of recto-vaginal fistulæ. Such ulcerative processes, which may cause strictures of the rectum, are sometimes due to syphilis, particularly in women, and are liable to be mistaken for cancer.

Clysmatic ulcers of the rectum are of mechanical origin.

Rectal fistulæ are divided into: complete, when the fistulous passage connects the lumen of the rectum with the surface of the perianal integument; external incomplete (blind), when the fistulous passage does not open into the lumen of the bowel; internal incomplete, when fistula springs from the mucous membrane of the rectum and does not open externally (fistulous deep ulcer of the rectum).

These fistulæ usually develop from small abscesses, more rarely, apparently, spontaneously; pretty often we find rectal fistulæ in pulmonary tuberculosis.

TUBERCULOSIS OF THE INTESTINE.

(Plate 45.)

A number of specific infectious processes is localized in the intestine; among these are typhoid fever (ileotyphus), cholera, dysentery, and tuberculosis.

The latter is rarely primary, more often secondary to pulmonary tuberculosis. While the former may be traced to alimentary infection (especially the ingestion of raw milk from tuberculous cows), the latter results from auto-infection, germ-laden sputa or their residues reaching the intestine with the food. In tuberculous children in whom cavities in the lungs are not frequent, ulcerative tuberculosis is much rarer (thirty to forty per cent) than in adults (sixty to seventy per cent). No lesion of the surface is necessary; the tuberculous virus is able to pass through the intact intestinal epithelium. The points of predilection for the arrest and multiplication of the tuberculous virus are the lymph follicles (Peyer's patches and solitary follicles) of the inferior small intestine and the adjoining colon, which have about the same predisposition to tuberculous infection as the other lymphatic glands of the body and the apices of the lungs. In the beginning of the affection we notice some follicles to be swollen to the size of a pin's head or even of a hempseed and of a dull yellowish color; pressure or incision shows that the follicle is changed to a soft cheesy mass. As the cheesy necrosing process gradually erodes the covering epithelial layer the centre breaks down and cup-shaped or crateriform ulcers appear, which rapidly enlarge by confluence in the region of Peyer's patches. In this way large losses of substance result, which have an eroded bottom and indented margins; corresponding to the course of the blood and lymph vessels they have a tendency to spread circularly and thus result transverse belt or ring shaped constricting ulcers (Plate 45), usually associated with slight stenosis of the affected portion of the intestine; at the bottom and margins yellowish nodules are frequently embedded. On the corresponding serous coat we often find the local ulceration of the mucosa sharply marked by a reddish, whitish, or somewhat slaty discoloration of the former, as well as by a local eruption of tubercles. As in the case of the lung, we can distinguish acutely developed and rapidly advancing ulcerations from those of gradual formation which enlarge slowly, in which case they show a slaty pigmentation at the bottom. Healing of the ulcerative defects is extremely rare, here and there we may find a secondary cicatricial stenosis. Besides the localized multiple and focal peritoneal tuberculosis there is frequently a diffuse tuberculosis of the peritoneum, with or without adhesion and agglutination of the different layers, and with or without effusion of a fluid or coagulated exudate.

Secondary cheesy tuberculosis of the mesenteric and retroperitoneal glands is often observed. Corresponding to the generally slow progress of the destructive process, perforation and perforation peritonitis are rather rare (about five per cent) in intestinal tuberculosis.

TYPHOID FEVER.

(Plate 46.)

The result of typhoid infection is a diffuse inflammation of the mucous membrane, together with a hyperplastic and necrosing inflammation of the lymphoid follicles of the lower ileum; frequently (about one-half of all cases) the solitary follicles of the adjoining colon are also involved (ileo-colonic form). At the same time a very similar inflammatory hyperplasia occurs in the mesenteric glands and in the spleen.

In the first stage (hyperplastic infiltration) we observe, along with diffuse catarrhal inflammation of the mucous membrane, great swelling and enlargement of the solitary follicles and of Peyer's patches; the former may reach the size of a hempseed or pea, the latter constitute flat, plane, reddened, elevated spots, projecting several millimetres above the level of the mucous membrane; on incision the proliferated gland tissue appears reddish-gray, and of semi-soft medullary consistence.

In the second stage (stage of cicatrization), which begins about the eighth to the tenth day, the swelling of the follicular glands begins to subside and the process recedes in mild cases, the glands acquiring a reticulated and often somewhat slaty gray appearance. Or else, in the typical course, with the tenth day begins the cicatrization of the infiltrations, that process frequently commencing at several points of the surface; the necrotic eschars, owing to the staining with bile, are of a dirty yellowish color and often fissured; the necrosing and diphtheroid process may also involve the submucosa and the muscular tunic.

In the third stage, which occupies about the third week, the eschars are cast off and ulcers form, having an oblong shape corresponding to the arrangement of the patches of Peyer, while tuberculous ulcers are more transversely placed and circular or ring-like in form. Owing to a demarcating inflammation, the eschars are first loosened, then cast off; the bottom and margin of the ulcer are irregular and jagged; erosion of vessels may occasion more or less profuse hemorrhage.

In the fourth stage (stage of recovery), whose duration varies and which occupies the fourth and fifth weeks, the ulcers clear and heal; the regenerative process starts from the margin and bottom of the ulcers and reproduces especially connective tissue and epithelium, but never causes the formation of cicatrices and stenosis of the bowel. A marked slaty pigmentation of the affected portions forms in the later stages of typhoid fever one of the residues of the above-described metamorphoses. When the course is normal, free from complications, the anatomical process in the intestine may have run its course at the end of a month.

Owing to the associated catarrhal inflammation of the bowel, the contents of the small intestine and colon are usually thin, offensive, and of the consistence and color of pea soup. A complication to be dreaded is perforation of the ulcers, which occurs in or after the third week and may cause fatal perforation peritonitis. In some cases the course is slow, so that even in the fifth, sixth, and seventh weeks open, slate-colored ulcers can be demonstrated; or else in the third week or later a relapse occurs (auto-reinfection). In rare cases a true gangrene of the intestine develops from the eschars, whose place is taken by irregular, friable, extremely fœtid masses. Septic and pyæmic mixed infection, starting from the eschars and ulcers, is at times superadded in all possible gradations. Partial eschar formation and purulent disintegration are sometimes found in the hyperplastic mesenteric glands and in the spleen. Among other complications occurring in typhoid fever more frequently than in any other infectious disease may be enumerated parenchymatous inflammation and fatty degeneration of the heart, liver, and kidneys, bronchitis, pneumonia, and bed-sores. In the majority of cases (sixty to seventy per cent) the fatal issue in typhoid fever is due to complications (mixed infection), in the minority to the intensity of the infection. As regards the time when death takes place, large statistics show that in one-half of all fatal cases it is in the third and fourth weeks, in twenty-five per cent in the second and fifth weeks, only in two and a half per cent in the first week, and in the remainder (more than twenty per cent) after the fifth week. The mortality varies greatly, in good hospitals it may be as low as eight to ten to twelve per cent, while formerly (thirty to forty years ago) it was as much as twenty-five per cent.

CHOLERA.

The morbid process is confined mainly to the small intestine.

In the first or algid stage the intestine is intensely reddened, of a rosy color; the wall is friable, greatly swollen, the mucous membrane in particular, owing to a serous infiltration. The follicles are generally swollen. The lumen contains a light gray, abundant fluid mixed with flakes and resembling rice water. As the biliary secretion of the liver is arrested, the fluid is almost colorless and odorless, of alkaline reaction and watery, containing only from one to two per cent of solids. The flakes consist of desquamated epithelial shreds (desquamative enteritis).

In the second stage (cholera typhoid) the intestinal contents are sparse and catarrhal alterations can still be demonstrated. Owing to the partial denudation of the mucosa there are a croupous exudation, eschar formation, and diphtheroid enteritis; transverse ulcers extending deep into the wall of the bowel are left behind. The admixture of bile again stains the contents a dark green, brown, or yellowish color.

During the first six or seven days comma bacilli are plentiful, most abundant in the lower portions of the ileum, and scattered here and there also in the duodenum and stomach. The average mortality is fifty per cent.

DYSENTERY.

The sporadic is to be distinguished from the acute epidemic form. The location of the disease, a hemorrhagic necrosing colitis and proctitis, is in the large intestine, the most intense changes being found in the rectum and the lower portion of the colon.

The mucous membrane is enormously swollen, almost fluctuating on touch, of a dark brownish-red color, and infiltrated with sero-cellular and hemorrhagic exudate. The contents of the bowel are usually mixed with blood and fœtid. Owing to the necrosis of the upper layers of the mucosa a branny deposit is rapidly formed: as the process advances the mucosa is discolored greenish-yellow or greenish-gray, especially on the acme of the folds. In the further course the eschars are cast off, leaving in the mucosa, which is sprinkled with slaty spots, irregular ulcers that eventually heal and produce cicatricial stenoses of the intestine.

In the tropics dysentery is far more frequent and severe than in temperate zones. The cause of tropical dysentery is said to be a protozoon (Amœba dysenteriæ), which, however, is always found to be associated with pathogenic bacteria.

Under the collective term of sporadic dysentery is understood an entire group of septic or toxic forms of colitis which present anatomical alterations similar to those of acute dysentery. This group includes diphtheroid, necrosing, and hemorrhagic forms such as occur in sepsis, uræmic auto-intoxication, mercurial poisoning, etc., in numerous gradations. Under the same head belongs follicular dysentery, which is characterized by multiple ulcers springing from the solitary follicles of the colon.

NEOPLASMS OF THE INTESTINE.

The most frequent new formation is cancer, whose point of predilection is the rectum (more than sixty per cent of all intestinal cancers); in thirty per cent the colon and cæcum are involved, the remaining four to seven per cent occur in the small intestine. Cancer of the bowel is most commonly met with between the fiftieth and sixtieth years of life.

Secondary cancer of the intestine occurs now and then, the primary neoplasm being in the pancreas or the stomach.

Anatomically the same forms are found in the intestine as in the stomach. Hard cancer (scirrhus) forms circumscribed nodules or a firm annular infiltration. A more frequent variety is medullary carcinoma which is characterized by its tendency to ulceration; now and then it takes the form of a papillary or villous tumor which develops from a polypoid adenoma. The least common is colloid cancer, which is met with mainly in the rectum. In the lowest portion of the rectum near the anus epithelioma is sometimes found.

As in the stomach, after the mucous membrane the submucosa, muscular tunic, and serous coat are early invaded; stenosis of the intestine occurs almost invariably, though it may again disappear when the superficial layers ulcerate and break down. As a result of the stenosis there are dilatation of the bowel above the tumor, sometimes associated with compensatory hypertrophy of the muscular tunic. Intestinal cancer often spreads to the peritoneum or to the neighboring organs, which are infiltrated by contiguity and destroyed. Thus cancer of the rectum may invade the vagina and uterus, the bladder in men (recto-vesical fistula), and the retroperitoneal pelvic connective tissue; secondary carcinosis of the mesenteric and retroperitoneal lymphatic glands and of the liver is almost invariably observed. More frequently than in gastric cancer perforation into the peritoneal cavity and terminal purulent perforation peritonitis come under observation.

PARASITES OF THE INTESTINE.

Aside from a few specific microparasitic organisms (tubercle, typhoid, and cholera bacilli), the great majority of vegetable occupants of the intestine belongs to the saprophytes which only occasionally assume pathogenic properties (facultative parasites), especially and most dangerously when they are enabled, by reason of nutritive, circulatory, or destructive influences, to penetrate into the tissue of the intestinal wall or into the peritoneal cavity.

For animal parasites the intestinal canal forms a special site of predilection.

Among the tape worms there are three species which find their habitat in the small intestine of man:

1. Tænia saginata (mediocanellata): head devoid

of hooklets, with sucking-discs; the large ripe segments contain numerous and finely branching uterine arborizations with ova. The undeveloped form of the parasite lives in the muscles of cattle.

2. Tænia solium: head provided with crown of hooklets and sucking-discs. The segments are less in breadth, width, and thickness than those of the preceding variety; the branches of the uterus are sparser and larger. The undeveloped form (cysticercus cellulosæ) lives in the muscles of the hog, exceptionally also in various organs (brain, eye, and muscles) of man, so that this parasite endangers health both in its mature and in its undeveloped condition.

3. Bothriocephalus latus: head almond-shaped, with flat sucking-discs; the segments are broader than they are long. The rosette-shaped uterus occupies the centre of the segments and is brownish in color. In water the ova change to a ciliated oncosphere which escapes from its shell and swims about. From this is developed, perhaps through the intervention of a secondary intermediate host, in fishes (particularly in the pike and in the burbot) a plerocercoid, 8–30 mm. long.

4. Ankylostomum duodenale (Dochmius duodenalis), 8–18 mm. in length. In the bell-shaped oral capsule are hook-like teeth. This blood-sucking parasite lives in the duodenum and upper jejunum, and produces grave anæmia (Egyptian chlorosis); it is the cause of the miner's disease or Gotthard anæmia, particularly in workmen occupied in tunnels, pits, and brickyards. The liberated ova change to larvæ which are very resistant to external influences,

and again find their way into the mouth with the water or through contaminated food or hands.

7. *Ascaris lumbricoides.* The male may reach 25 cm., the female 40 cm. in length. Habitat, in the small intestine. It is one of the most common parasites, particularly in children, and is more frequent in the country than in cities. The worms may be present in small or large numbers. Under favorable conditions (moisture and heat) the fertilized ova develop to the embryonic stage in from four to six weeks, and partly developed ova reach the digestive tract with the drinking-water or direct from the ground and attain their maturity in the body.

8. *Oxyuris vermicularis* (thread or seat worm). The male may reach 4 mm., the female, which is far more numerous, 10 mm. in length. These worms are very frequent in children and adults, in the cæcum and adjoining colon; they pass with the feces into the lower rectum, leave the anus especially during rest in bed, and cause annoying sensations. The ova reach the intestinal canal by adhering to the fingers or food substances (raw fruit); auto-infection is very frequent.

9. *Trichocephalus dispar* (whip worm), 4–5 cm. in length. The thread-like anterior extremity (⅗ of the entire length) is joined to a thick posterior extremity. The parasite is present in small numbers in the cæcum and colon. Infection is direct without an intermediate host by the ingestion of ova in the embryonal stage. Only exceptionally does it cause any symptoms.

10. *Trichina spiralis.* Male 1.5 mm., female 3–4 mm. in length. Habitat, the small intestine of man

and many mammals. The muscle trichinæ introduced with the food (pork) escape from their capsule, rapidly attain maturity, and copulate; the females, which may live seven weeks, bear living young (0.1 mm. long) which pass through the intestinal wall and change to muscle trichinæ in the muscles. Hogs acquire their trichinæ, as a rule, from rats, in which they are extremely frequent, now and then also from other hogs (feeding with slaughter-house offal).

ABNORMAL POSITION OF THE INTESTINE.

Under this head belong herniæ, invaginations, volvulus, and prolapse.

Hernia.

By hernia (rupture) we understand the position of a portion of the intestine outside of the abdominal cavity; the covering, the hernial sac, consists of the protruding parietal peritoneum and the external integument (cutis and subcutis). We distinguish external and internal herniæ.

In external hernia (the most frequent variety) the sac inclosing the prolapsed intestine consists of a sacculated protrusion of the parietal peritoneum within the accessory coverings (cutis and subcutis). In every hernia we must distinguish the hernial opening, the neck, the body, and the contents of the hernial sac.

The majority of herniæ are acquired anomalies in the occurrence of which several factors may be active. Predisposing causes are inherited tendency, shape of the abdomen, inclination of the pelvis, and local proliferation of knuckles of fat in the subserosa of the parietal peritoneum, whereby the fibrous parts are crowded apart and the affected portion of the peritoneum is dragged outward. Abnormal suspension of the intestines, especially lengthening of the mesentery (enteroptosis), has also a predisposing effect. The exciting cause acts as follows: forcible straining, at once or when repeated, raises the intra-abdominal pressure, the less resistant portions of the wall give way, and local protrusion results.

Congenital herniæ (only inguinal and umbilical herniæ) are among the arrests of development and represent persistent diverticula of the peritoneum.

The most important forms are:

1. Inguinal hernia, constituting about five-sixths of all herniæ, is found in men ten times more frequently than in women. A congenital inguinal hernia consists of the open processus vaginalis peritonæi, which is changed to a hernial sac when intestines prolapse into it; the contents (intestine) are in direct contact with the testicle; the hernial opening begins laterally from the epigastric artery ("external" inguinal hernia).

Much rarer than the external (lateral) inguinal hernia is the internal variety situated medially from the epigastric artery; this hernia passes vertically through the abdominal wall.

2. Crural hernia is far less frequent than inguinal hernia (1 : 9), and affects women more often than men (3 : 1). The hernial sac, which is small, protrudes from under Poupart's ligament, crowding before it the fibres of the fascia cribrosa.

3. Umbilical hernia (three per cent of all herniæ) occurs congenitally as an arrest of development with

fissured abdomen; the prolapsed intestine is covered only with the external layer of the funis. Acquired umbilical hernia is most frequent in corpulent persons and in women who have borne many children. Quite an extensive variety is hernia of the umbilical cord, the funis dilating and receiving intestines when the abdomen is fissured.

Among the rare forms are sciatic hernia (protrusion of the peritoneum through the sciatic notch) and hernia through the obturator or oval foramen. Diaphragmatic hernia is not a true hernia but a prolapse of abdominal contents into the left pleural cavity through a defect in the diaphragm; it may be congenital or acquired after a trauma (fall from a height). Internal herniæ so-called (Treitz's hernia) are characterized by the fact that the intestine is displaced through preformed pockets of the peritoneal wall. Such retroperitoneal herniæ occur very rarely, for instance, at the foramen of Winslow, in the subcæcal fossa.

A hernia of the intestinal wall (Littré's hernia) is present when only one side of the intestinal wall lies in the hernial sac as a diverticular protrusion.

Herniæ are among the frequent affections; two and a half to three per cent of all persons are suffering from them; by reason of numerous exciting causes they are found more than four times as often in men as in women. Herniæ are more frequent on the right than on the left side; sometimes they are bilateral.

The hernial contents vary greatly in bulk and anatomical character; besides the intestine with the corresponding mesentery we often find portions of the great omentum which are rich in fat, almost lipomatous. The prolapsed parts are either freely movable (reducible) or adherent to the peritoneal investment of the sac (irreducible).

The most serious complication of a hernia is incarceration with occlusion or stenosis of the intestine (different degrees of stenosis).

The term elastic incarceration is employed when, the hernial opening being relatively narrow, a sudden increase of the intra-abdominal pressure (forced inspiration, sneezing, straining) crowds a portion of the intestine into the sac and reduction is prevented by the elastic constricting opening. In fæcal incarceration the cause lies in the intestine itself; this variety begins with the arrest of the contents, the expanding bowel drags adjoining loops toward it, and the patulousness of the afferent and efferent loops is suspended. The occlusion of the intestine causes the symptoms of ileus (miserere; stercoraceous vomiting), dilatation of the superior portions of the bowel, and meteorism. As a result of the compression exerted by the elastic hernial opening, which occludes particularly the efferent veins of the mesentery, there are venous hyperæmia, hemorrhagic œdema, and hemorrhagic dropsy of the hernial sac (sanguineous appearance of the hernial fluid), and finally gangrene and perforation of the constricted intestine, with septic peritonitis.

Among other complications may be enumerated simple coprostasis, which may increase to fæcal incarceration and inflammation in numerous forms which may cause diffuse peritonitis.

Invagination (intussusception) is the entrance of a portion of the intestine into the adjoining inferior portion; the homonymous layers of the intestinal wall then touch each other. The invagination involves also the mesentery or the mesocolon; venous hyperæmia, stasis, hemorrhage, and hemorrhagic infarction result; the invaginated portion forms a slightly curved, sausage-shaped, dark brownish-red mass. The intussusceptum consists of a descending inner cylinder and the ascending middle cylinder; the external (likewise descending) covering or sheath is formed by the inferior segment of intestine. In consequence of the occlusion the results are the same as in incarceration, *i.e.*, dilatation of the superior portions of the bowel. The most frequent variety is the ileo-cæcal, ileum and cæcum being invaginated into the colon; more rarely we find invaginations of the ileum or colon. Agonal invaginations of the intestine are frequent in infants; they are sometimes multiple, easily reduced, and free from every secondary reaction.

Volvulus is a torsion of the intestine either around its longitudinal axis, rarely observed in the cæcum or colon, or the mesentery or mesocolon is twisted, the fixed point being at the origin of the mesentery; intestinal occlusion occurs secondarily. There may also occur internal incarcerations with stenosis or occlusion of the intestine by pseudo-ligaments, synechiæ, especially between the great omentum and pelvic peritoneum, and by fissures in the omentum and mesentery.

Prolapse is the protrusion of intestine through the anus, *i.e.*, invagination of the bowel at its external opening. The surface of the prolapsed intestine consists of mucous membrane. This anomaly develops under excessive and oft-repeated straining, in relaxation of the intestine and when it is adherent in the true pelvis. Secondarily inflammatory processes and gangrene may result.

DISEASES OF THE PERITONEUM.

The peritoneum covers numerous and bulky organs; among the serous membranes of the body it possesses by far the greatest surface; this fact on the one hand, and the frequency of diseases of the organs which it covers on the other hand (stomach, intestinal tract, female genitals), make it easy to understand that changes of the peritoneum, especially such as are secondary in their nature, are observed in numerous instances.

Ascites is met with either as a local symptom of general dropsy or occurs by itself in connection with prolonged and marked congestive hyperæmia in the course of the portal vein, usually due to cirrhosis, carcinoma, and syphilis of the liver (hæmatogenous mechanical ascites), or local diseases of the peritoneum, particularly neoplasms (carcinosis, sarcomatosis), cause disturbed absorption and dropsy. In some cases several causes are combined, *e.g.*, hydræmia and circulatory disturbances, or inflammation supervenes when dropsy has existed some shorter or longer time, *i.e.*, inflammatory ascites. The quantity of fluid is very variable; it ranges from a few tablespoonfuls to ten and twenty quarts.

Often we find as co-ordinated associated disturbances congestive catarrhs of the gastro-intestinal mucous membrane, œdema of the intestinal wall, and congestive enlargement of the spleen. The sequelæ depend in the main upon the quantity of the transudation; when the amount of fluid is large, we observe abnormal elevation of the diaphragm with corresponding diminution of the thoracic space and of the organs contained therein, especially the lungs.

Peritonitis is extremely frequent in all gradations and forms—circumscribed or diffuse; acute or chronic; benign, tending toward adhesion and recovery, or as an infectious and purulent process with a usually fatal termination.

As a rule peritonitis is a secondary process springing from the abdominal organs; very rarely is it idiopathic and cryptogenic.

Adhesive peritonitis is not often diffuse but more commonly develops from dry fibrinous peritonitis. Such circumscribed forms of adhesive peritonitis are found particularly in the true pelvis (adhesive pelvic peritonitis), *i.e.*, as perimetritis, peri-oöphoritis, perihepatitis, perisplenitis, perityphlitis, or as adhesion of the gall bladder to the adjoining organs.

Exudative peritonitis may be diffuse or encapsulated. In general the liquid masses of exudate have the tendency to collect in the deeper portions of the abdominal cavity, especially in the true pelvis, in the neighborhood of the kidneys, the liver, and the spleen. Besides the fibrinous and sero-fibrinous form, which has a more benign character, we distinguish a hemorrhagic, a purulent, a fibrino-purulent, an ichorous, and a tuberculous peritonitis.

The gross and the minute alterations in the forms of peritonitis here enumerated agree in general with the analogous processes in other serous membranes (pleurisy, pericarditis) described above. The quantity of the exudate varies according to the duration and the intensity of the process. The amount may be minimal and still cause death; in other cases we may find several quarts of an inflammatory exudate, especially in hemorrhagic and sero-fibrinous peritonitis. In the neighborhood of the starting-point, for instance, in perforation peritonitis, the exudation is usually most abundant. The more recent the inflammation the more commonly are the organs simply adherent and easily liberated; when the process is of long standing the adhesions are firm.

Among the secondary alterations accompanying peritonitis the most important to be enumerated are inflammatory œdema of the intestinal wall, meteorism due to paralysis of the intestinal muscles, and elevation of the diaphragm; encapsulated purulent exudates may occasionally perforate into the intestine and recovery may thus be favored. When this occurs, the loss of substance at the point of perforation is usually greater in the serosa and subserosa than on the mucous surface.

In perforation peritonitis starting from the intestinal tract gases may escape into the peritoneal cavity and cause tympanites; in ichorous forms of peritonitis resulting from the escape of intestinal contents the development of gas may also take place subsequently from the exudate.

Ichorous and sanio-purulent peritonitis arises particularly after perforation and injuries of the intestine (vermiform appendix) and in incarcerated hernia. The fœtid exudate which is mixed with fæces contains bacteria of decomposition.

Etiologically it may be noted that in intestinal perforation peritonitis sometimes minute quantities of the contents of the bowel entering the peritoneal cavity may suffice to set up a fatal peritonitis; of course such small amounts may also be absorbed or encapsulated. As a rule in perforation peritonitis there is a multiple or mixed infection (in which the bacterium coli in its numerous varieties usually takes part), while the hæmatogenous and metastatic forms of peritonitis generally represent mono-infections (with streptococci or staphylococci). The contents of the colon are more dangerous than those of the small intestine. A peritonitis of aseptic origin caused by chemical irritants (*e.g.*, bile, or a filtration of fæces free from bacteria) furnishes no purulent but a serofibrinous and hemorrhagic exudate. In perforation peritonitis the peritoneal cavity is invaded not only by pathogenic bacteria but also by chemical products (toxins, proteins, intestinal ferments, and products of fermentation) which are apt to influence the secondary peritonitis. It is not yet positively determined whether death in infectious peritonitis is due to intoxication or to nervous influences (continual nerve irritation, *i.e.*, slowly acting shock).

Tuberculosis of the Peritoneum.

(Plates 48, 49, and 50.)

Tuberculosis of the peritoneum occurs in several forms:

1. As acute miliary tuberculosis, either of hæmatogenous origin and a local phenomenon of acute general miliary tuberculosis, or by continuity from

local tuberculosis of the intestinal mucosa or of the mesenteric and retroperitoneal lymphatic glands (Plate 49). It is usually spread more or less uniformly over the entire peritoneum, the slightly reddened serosa being covered with numerous miliary tubercles; when the course is less rapid (subacute or subchronic tuberculosis) the nodules may reach the size of a hempseed or pea.

2. As localized focal miliary tuberculosis, frequently associated with subacute or chronic fibrous peritonitis, starting from older tuberculous ulcers of the intestinal mucosa. Particularly in the serosa of the lower ileum and the adjoining colon we observe upon the convex side of the slightly contracted bowel groups of miliary nodules, each group corresponding to an ulcer on the mucous surface of the intestine, *i.e.*, aggregated recurrent miliary tuberculosis resulting from regional infection (Plate 48).

3. Tuberculous peritonitis may resemble tuberculous pleurisy; we distinguish an acute or subacute exudative form, when the exudate is usually hemorrhagic, more rarely sero-fibrinous or purulent; besides, numerous miliary tubercles like grains of sand are scattered through the serosa. Then there is a chronic productive and adhesive tuberculous peritonitis with the formation of larger nodules (Plate 50), diffuse infiltration especially of the great omentum (omentitis tuberculosa), which is shortened, fibrous, thickened, hard as a board, and sprinkled with irregular cheesy foci.

Tuberculosis of the peritoneum, with or without inflammation, is found in about five per cent of all cases of fatal tuberculosis; very rarely is it primary, generally secondary. In men it is remarkably more frequent than in women (4 : 1); this predisposition in men probably depends upon the corresponding frequency of hepatic diseases and alcoholism. Spontaneous recovery from peritoneal tuberculosis is quite rare; in recent years arrest of the disease and even recovery have been reported after simple laparotomy with removal of the liquid and coagulated masses of exudate.

Neoplasms of the peritoneum are usually secondary; most frequently we observe secondary carcinosis in the form of multiple nodules and tubercles (Plate 51) or diffuse infiltrations of the omentum and mesentery with primary cancer of the stomach, intestine, liver, or pancreas. Ascites or states of inflammatory irritation are often associated with it.

Among primary neoplasms of the peritoneum and its subserosa may be enumerated sarcoma, endothelioma, rarely fibroma and lipoma, also myoma which may spring from neighboring organs containing muscular tissue (subserous myoma of the uterus and its appendages).

Of animal parasites echinococcus cysts and rarely cysticercus cellulosæ are found.

Enteroptosis (Glénard's disease) is the term applied to anomalous position of the abdominal viscera characterized chiefly by elongation and stretching of the suspensory ligaments of the stomach and colon. As a result of this condition we find these organs abnormally movable and in faulty positions, *e.g.*, the transverse colon, especially its right flexure, is situated at the level of the umbilicus or lower, midway between symphysis and umbilicus; the stomach (gastroptosis), too, is correspondingly displaced downward and to the left. Descent of the colon is caused not only by abnormal stretching of its ligaments but also by long-continued or oft-repeated overfilling. Slighter grades of enteroptosis are physiological and give rise to no disturbances. Unsuitable clothing (tight lacing), frequent pregnancy, and former obesity are evidently important etiological factors.

Diseases of the Liver.

Injuries of the liver are met with as gunshot, incised, and stab wounds, and as subcutaneous ruptures, the latter resulting especially from blunt force or a fall from a height; they end in death by fatal hemorrhage into the abdominal cavity or by secondary infectious inflammation. The escape of bile into the abdomen causes sero-fibrinous peritonitis and sometimes terminates fatally.

The capsule of the liver takes part in the diffuse inflammations of the peritoneum and in numerous, particularly inflammatory processes of the liver; in this way, for instance, arises in hepatic cirrhosis a chronic adhesive and hyperplastic inflammation which very rarely may also be observed idiopathically with secondary atrophy of the liver.

Among the circulatory disturbances of the liver should be mentioned as a frequent form chronic congestive hyperæmia (Plate 52, Figs. *a* and *b*). This occurs regularly when the return of venous blood from the liver is retarded by valvular and muscular diseases of the heart. In the initial stages the liver is enlarged and of a dark cyanotic color; when of long standing there is capillary ectasia in the centre of the acini surrounding the central veins, with secondary atrophy of the liver cells. The centres of acini in consequence present a dark brown to blackish-red color, while the marginal portions of the acini are light gray or pale yellowish. This alternation of dark spots and light reticulated tints produces a very characteristic mottling of the cut surface (nutmeg liver), which, according to the amount of blood and the associated or absent fatty infiltration of the marginal zones of the acini, shows various gradations —dark and light (anæmic) shades of nutmeg liver. When the circulatory disturbance is of long standing the entire organ appears shrunken (atrophic nutmeg liver, cyanotic or red atrophy).

ATROPHY OF THE LIVER.

Partial atrophy of the liver is frequently found in the so-called corset liver, in which the furrow, varying in width and depth, runs transversely across the antero-inferior portions of the right and sometimes also of the left lobe.

Simple diffuse atrophy occurs in very old, cachectic, or greatly emaciated patients; the bulk of the liver may sink to half of the normal; the tissue is firm and of a dull brown color (brown atrophy); a finely granular pigment is found deposited in the centre of the acini.

Acute yellow atrophy of the liver (Plate 54), so called, belongs to the group of degenerative and necrotic processes and will be discussed with degeneration of the liver.

HYPERTROPHY OF THE LIVER.

Partial vicarious hypertrophy (regeneration) of isolated segments is found sometimes after the loss of portions of the liver, in a specially characteristic form in echinococcus and syphilis, or in the lower segments of the right lobe after pronounced pressure atrophy (corset liver) of the adjoining portions.

Diffuse true hypertrophy occurs occasionally, especially in connection with slight increase of the supporting framework—a condition which is largely but erroneously included with hypertrophic cirrhosis (pseudo-hypertrophy); the etiological factors are chronic congestion, plethora, and the abuse of alcohol (beer).

FATTY LIVER.

An abnormal accumulation of fat in the liver is produced in different ways.

1. Fatty degeneration develops rapidly in connection with and as a second stage of cloudy swelling (parenchymatous hepatitis). The liver is moderately enlarged, pale, and succulent; in the liver cells we find numerous droplets of fat, usually of small size. In the higher grades (*e.g.*, in acute intoxications) the liver is enlarged from thirty-three to fifty per cent, is of a gray clayey color, sometimes icteric, and the liver cells are completely filled with drops of fat. This acute fatty degeneration of the liver is found in moderate degree in grave acute infectious diseases (septic or pyæmic infections, typhoid fever) associated with high pyrexia; besides it develops in a few days with intoxications (poisoning by phosphorus, arsenic, and iodoform). The highest degrees of fatty liver combined with icterus are found in phosphorus poisoning (the contained fat rises from three per cent to thirty-two per cent of the fresh substance); the fat accumulating in large masses in the liver is formed

only to a slight extent from the protoplasm of the liver cells (fatty degeneration); the greater portion is carried to the liver from other organs which have undergone a similar degeneration (fatty infiltration).

In acute yellow atrophy, a process which for the time is still obscure, a considerable portion of the hepatic parenchyma perishes by fatty necrosis; the liver diminishes markedly in size within two to three weeks and appears flabby; on the cut surface red (alcoholic) and yellow (icteric) portions alternate (Plate 54).

2. The second principal form of fatty liver (fatty infiltration, Plate 52, Fig. *a*) is characterized by the fact that the fat is deposited in larger drops in the otherwise intact, merely enlarged liver cells; whether by reason of increased supply, or of diminished combustion, or of both combined. Belonging in part still among the normal conditions is the fatty liver of overfeeding which is met with in obesity; in the higher and extreme degrees (the contained fat rising to forty-four per cent of the fresh substance) it is pathological, as for instance in the artificially produced fatty liver in overfed geese. Another form of this fatty infiltrated liver is frequently found in drunkards, sometimes associated with slight or marked degrees of cirrhosis (fatty cirrhosis).

3. Finally, a pronounced fatty infiltration of the liver is sometimes met with in very cachectic and emaciated persons (cachectic fatty liver), especially in cases of phthisis, carcinoma, and chronic fevers. The origin of this anomaly is rather obscure; perhaps the associated anæmia, increased decomposition of albumin, and consequent decreased combustion of

fat have something to do with it; conditions resulting from infection may sometimes also have their share. In all these forms the liver is markedly enlarged, the sharp margins are rounded, the tissue is cut easily, its consistence is almost like that of butter, and it is bloodless. Sometimes slight icterus is present, evidently the result of stenosis and occlusion of the smaller bile ducts by the excessively swollen and fatty infiltrated parenchyma of the liver.

Inflammation of the Liver. Hepatitis.

Purulent hepatitis is always focal and usually multiple; the abscesses vary in size and may reach that of a fist and larger; the pus is often mixed with detritus, shreds of tissue, and bile. The disease is generally of embolic origin, springing from the radicles of the portal vein when infectious, purulent, and septic processes have developed in the neighborhood of the branches of that vessel, especially in the wall of the colon (dysentery) or the vermiform appendix, and caused septic thrombo-phlebitis. The abscesses are usually subcapsular and may give rise to purulent perihepatitis and peritonitis. The portal branches of the liver are often filled at the same time with discolored, semi-soft thrombi undergoing purulent disintegration. The hepatic abscesses which are so frequent in the tropics and were formerly regarded as idiopathic affections are probably without exception of embolic origin and develop by preference in connection with tropical dysentery. By paradoxical or retrograde embolism infected thrombi, perhaps derived from the peripheral veins of the head, may penetrate from the right auricle into the inferior vena cava and into the hepatic veins and thus produce abscesses of the liver.

CHRONIC INTERSTITIAL HEPATITIS. CIRRHOSIS OF THE LIVER.

Plate [illegible]. Fig. 1.

This occurs in two principal forms:

1. Atrophic cirrhosis, granular cirrhosis (Laennec). The liver is markedly diminished in size, the surface is granulated with coarse and fine tuberosities, the color is yellowish or pale grayish-yellow, and the tissue creaks under the knife. On the cut surface we notice that the sustentacular tissue is irregularly and profusely proliferated; between the whitish masses of connective tissue appear the irregularly shrunken acini, which are more intensely yellow or icteric in color and often infiltrated with fat. The microscope reveals a periportal interacinous connective-tissue proliferation. Owing to the obliteration of branches of the portal vein there is a secondary stasis in the distribution of that vessel, *i.e.*, congestive enlargement of the spleen, chronic catarrh of the stomach and intestine, and finally ascites; the obliteration and constriction of bile ducts cause moderate icterus.

2. More rarely we observe hypertrophic cirrhosis of the liver (pseudo-hypertrophy), the organ being frequently enlarged to one and two-thirds of the normal, sometimes to double and treble the usual bulk. The surface with few exceptions is smooth, the tissue usually richer in blood than normal; under

the microscope we notice in typical cases a moderate interacinous, together with a predominant intra-acinous, irregular proliferation of connective tissue; icterus is usually absent. In some cases a chronic icterus develops gradually and progressively for many months and even years to a fatal issue.

In other cases with hypertrophic induration of the liver the connective-tissue proliferation is so slight that it will hardly explain the marked enlargement of the organ. In such cases, which present no other complication (congestion in the distribution of the portal vein), the condition is evidently a true hypertrophy of the parenchyma with slight interstitial proliferation. Hypertrophic cirrhosis of the liver, which very rarely only is also found as the first stage of atrophic cirrhosis, occurs chiefly in men (ninety per cent of all cases) addicted to beer drinking (alcoholism). Often it constitutes an incidental condition, particularly in idiopathic cardiac hypertrophy and in tuberculosis; while a secondary splenic tumor is constantly found as a concomitant phenomenon, icterus occurs in only about one-fifth of all cases, and ascites is quite exceptional.

The various forms of hepatic cirrhosis are frequently associated with moderate and high degrees of fatty infiltration; besides there is sometimes an adenoid proliferation of the bile ducts in the hyperplastic interacinous connective tissue. In the etiology of chronic interstitial hepatitis toxic and infectious influences, especially alcoholism and syphilis, play a prominent part.

A special though rare form of hepatic inflammation is biliary cirrhosis which develops in connection with long-continued stasis of the bile and chronic inflammation of the bile ducts, *e.g.*, experimentally after ligation of the ductus choledochus. Under the same head belongs also verminous cirrhosis of the liver in domestic animals (sheep and cattle) caused by distomatosis; the morbid process is usually spread irregularly in spots throughout the liver; the marginal portions are as a rule more markedly affected than the central parts of the organ.

Tuberculosis of the liver may occur as acute miliary tuberculosis; in the parenchyma and visible through the capsule we find more or less numerous gray, punctiform to pin-head-sized nodules (Plate 56). This form of tuberculosis is more frequent in children as a local phenomenon of general miliary tuberculosis; now and then it is seen as the expression of the general terminal infection in chronic pulmonary tuberculosis of adults. More rarely the liver contains larger tuberculous foci, so-called solitary tubercles the size of a hempseed or pea, which are usually present in considerable numbers in the parenchyma of the organ. As a result of central softening and liquefaction small cavities form, the contents of which may be bile stained when there is communication with bile ducts.

The most important animal parasite of the liver is the echinococcus, the early life stage of a small tapeworm with three segments infesting the dog, tænia echinococcus (Plate 55, Fig. *b*). Dogs acquire the corresponding scolices from domestic animals (sheep, cattle, or hogs), while the ova of the dog's tænia in turn infect the latter and man. The parasite forms cysts which take the place of the liver tissue and are usually large, reaching the size of an adult head. Each consists of a mother cyst with chitin layer, a parenchyma layer upon which the scolices are situated, and generally contains numerous daughter cysts, *i.e.*, endogenous echinococcus. A special variety is the multilocular or alveolar echinococcus, which forms tumid masses of board-like hardness the size of a child's or even an adult head; in the centre is usually a cavity with eroded walls. A fibrous framework with few cells contains the folded and irregularly distributed chitin vesicles with or without scolices.

SYPHILIS OF THE LIVER.

(Plates 57, 58, and 59.)

Aside from diffuse interstitial hepatitis which sometimes develops in consequence of syphilis, either soon after infection or many years later as an after-effect, and is met with especially as the hypertrophic form in congenital syphilis of the newly born, syphilis occasionally gives rise to a serious visceral disease, a peculiar form of interstitial hepatitis, in which the proliferation of the fibrous tissue is so localized that the liver is markedly deformed (hepatitis interstitialis deformans). Thus irregular larger and smaller lobes are formed (Plate 57), which are separated by deep indentations and fissures (large-lobed liver). Very often these firm fibrous trabeculæ spring from the suspensory ligament; this frequently contains scattered yellowish, necrotic, firmly elastic nodules (gummata), *i.e.*, interstitial gummous hepatitis (Plates 58 and 59). The partial inflammatory atrophy often leads to compensatory irregular hypertrophy of neighboring segments. The liver as a whole, however, is always diminished in size; its characteristic shape is destroyed, the sharp margins are usually blunted, the capsule is covered with connective-tissue adhesions (perihepatitis adhaesiva). Owing to the connective-tissue sclerosis, especially at the porta of the liver and in the neighborhood of the branches of the portal vein, there are generally the same circulatory disturbances as in atrophic cirrhosis, namely, ascites, splenic tumor, and congestive catarrh of the gastro-intestinal mucous membrane.

NEOPLASMS OF THE LIVER.

Plate 60.

Of occasional occurrence in the liver are cavernous angioma, very rarely sarcoma (hemorrhagic angiosarcoma), and adenoma.

By far the most frequent neoplasm is carcinoma, which is only exceptionally primary, but occurs more often than in any other organ secondarily and metastatically.

Primary carcinoma of the liver forms large tubercles (from the size of a fist to that of a child's head) around which smaller nodules are visible; besides the tubercular form infiltrated carcinoma is met with, when the tissue contains insular cancer nests resembling the residues of liver tissue in cirrhosis of the organ.

Metastatic cancer occurs in the form of multiple tubercles which are usually almost uniformly distributed through the parenchyma and subperitoneally, and is especially frequent in primary carcinoma of the stomach, intestine, female genitals, breast, and external skin. The tubercles range in size from a pin's head to a fist, contain a central depression on the surface (umbilication), are sharply demarcated from the neighborhood, and whitish in color. Icterus and fatty infiltration of the hepatic parenchyma are sometimes found as complications.

Secondary cancer of the liver also develops occasionally by direct extension from the stomach (lesser curvature or pylorus) or from the gall bladder and the larger bile ducts.

DISEASES OF THE BILE DUCTS.

The biliary passages of the liver, especially the finer ones, participate in the diseases of the hepatic tissue; especially with interstitial inflammation of the liver, with diffuse parenchymatous processes, there may be stenosis or occlusion of the lumen with prevention of the escape of the bile, *i.e.*, icterus.

Inflammation of the bile ducts—cholangitis—develops along the ductus choledochus by extension from the duodenum; swelling of the mucosa and increased secretion (formation of a mucous plug) are said to prevent the escape of the bile into the intestine and cause catarrhal icterus, so called. Chronic catarrhs are due mainly to gall stones; these latter occasionally produce in the ductus choledochus circumscribed necrosing inflammations (pressure necrosis) which may result in perforation and secondary peritonitis.

Purulent inflammation of the bile ducts develops at times in connection with gall stones and with perforation of hepatic abscesses into the ducts; the latter contain bile-stained pus mixed with desquamated epithelia.

Dilatation of the bile ducts, especially the larger, occurs in connection with stenosis or obliteration of the ductus choledochus; inflammation of the mucosa is very apt to be associated with it when the escape of the bile is prevented. When the occlusion of the ductus choledochus has lasted for a certain length of time, there will be considerable dilatation of the bile ducts and of the gall bladder as well; rarely the latter may rupture.

When the cystic duct is occluded (for instance, by gall stones) the gall bladder is usually dilated and filled with mucus or watery fluid, *i.e.*, dropsy of the gall bladder by retention.

Inflammation of the mucosa of the gall bladder (cholecystitis) occurs in different gradations, namely, as simple catarrh with increased secretion of mucus and as purulent inflammation, particularly in consequence of gall stones. Necrosing (diphtheroid) and ulcerative cholecystitis with perforation, peritonitis, or evacuation into the stomach, duodenum, or colon may occur in typhoid fever. Very often we find adhesive pericholecystitis (agglutination with the transverse colon) as a residue of former inflammatory processes.

Inflammation of the gall bladder may also cause its atrophy and obliteration, especially in cholelithiasis (cholecystitis calculosa), which is, as a rule, associated with obliteration of the cystic duct.

When the obstruction to the outflow of bile persists for a greater length of time it will cause enlargement of the liver with pronounced icterus of the latter (icterus viridis), which presents a dark olive-green color (Plate 53); occasionally we observe biliary cirrhosis. The causes of the chronic stasis icterus are numerous; aside from those mentioned (inflammation and calculi) we may name tumors of the liver, especially carcinoma, gummata, and echinococcus, also tumors at the porta of the liver (carcinoma of the portal lymphatic glands, cancer of the pancreas, and cancer of the duodenum).

GALL STONES.

These differ greatly in size, form, and composition. They range in size from that of a hempseed to that of a small hen's egg; they are usually spherical or roundish or oval, representing true casts of the gall bladder; when numerous they are often facetted, polygonal, or cuboid. The principal forms to be distinguished are:

1. Cholesterin stone, usually roundish or oval, whitish or pale yellowish in color, of a dull lustre, the fractured surface radiating.
2. Cholesterin and bile-pigment stones; these consist of cholesterin and bile pigment, are usually yellowish-brown to deep black in color and of most frequent occurrence.
3. Pigment and calcium stones, consisting of bile pigment and calcium, sometimes lamellated; rather rare.
4. Calcium-carbonate stones, of whitish or grayish-white color; very rare.

In the formation of gall stones several different

[illegible]

[illegible] of women suffer- [illegible] are affected with [illegible] cases gall stones are [illegible] they having [illegible] the patient. As a rule they [illegible] until they migrate, whether [illegible] passages of the biliary sys- [illegible] and destruction [illegible] after the [illegible] the escape of stones [illegible] into the stom- ach and [illegible] gall stones may pass through external or biliary fistulæ which open on the skin of the abdomen; in this way gall stones may escape spontaneously into the outer world.

New formations in the gall bladder are not common; papillary epithelioma (cancer of the gall bladder) occurs sometimes, especially in women, and extends by contiguity to the neighboring liver tissue. Almost without exception gall stones are found firmly impacted in the cancerous mass, forming as it were the nucleus of the carcinomatous proliferation; gall stones evidently favor the development of cancer in predisposed subjects. Cancer springing from the smaller bile ducts branches dichotomously along Glisson's capsule and penetrates into the liver like an infiltration.

ICTERUS, JAUNDICE, CHOLÆMIA.

(Plates 53 and 54.)

By icterus we understand the pathological condition in which constituents of the bile, especially bile pigment, are present in the blood and in the tissues of the body.

Mechanical, absorption, or stasis icterus develops when obstructions in the bile ducts, from the ductus choledochus to the smallest ramifications in the liver, partly or wholly prevent the physiological escape of the bile. Under this head belongs icterus occurring in connection with stones in the ductus choledochus, with tumors, and with parasites. When the flow of bile is obstructed icterus occurs after the lapse of thirty-six hours. Catarrh of the duodenum with extension to the ductus choledochus is said to be sufficient to produce one of the most frequent forms, so-called catarrhal icterus. In various forms of hepatic cirrhosis, in interstitial syphilitic hepatitis, compression or obliteration of the smallest bile ducts in the liver takes place, with consequent escape of the bile into the blood. Changes in the blood pressure after birth and patency of the ductus Arantii are said to cause icterus neonatorum.

Through the influence of infectious or toxic materials which destroy the blood corpuscles—for instance, in sepsis, septico-pyæmia, pneumonia, poisoning by phosphorus, arseniuretted hydrogen, pyrogallic acid, ether, chloroform, mushrooms, snake bite, and toluylendiamin—an icterus occurs without the occlusion of the bile ducts, that was formerly enumerated as the opposite to the hepatogenous or mechanical form. The theory that the free hæmoglobin in the blood could without the intervention of the liver be directly transformed into bile pigment, was rendered improbable by the fact among others that in this form of icterus biliary acids could be demonstrated in the urine. The theory of hæmatogenous icterus was definitively disposed of by experiments on birds, whose liver was placed out of function by ligating the ductus choledochus and the blood-vessels, when it became impossible to produce icterus by the poisons named above. Without the liver there is no bile pigment and hence no icterus. These infectious and toxic forms of icterus, therefore, are primarily dependent upon extensive blood alteration (disintegration of many red corpuscles) and have more properly been designated as hæmato-hepatogenous icterus. Such an icterus with polycholia, accordingly, results when the liberated hæmoglobin as it were overwhelms the liver with abundant material for bile formation, and this being formed in abnormally large amounts escapes into the blood. In some varieties of toxic icterus (*e.g.*, in phosphorus poisoning) the changes in the liver (swelling of the cellular elements, especially the epithelia of the bile ducts) suffice to explain the disease in a purely mechanical way. Circulatory disturbances in the liver—reduced pressure in the hepatic vessels (capillaries) below that in the bile ducts—are sufficient to cause icterus (this probably includes the variety occurring in the newly born). Chronic venous hyperæmia of the liver, such as is present in nutmeg liver, likewise produces a slight degree of icterus.

Whether icterus (nervous form) can result from emotional excitement (anger) or from spasmodic contraction of the ductus choledochus (spasmodic icterus,) *e.g.*, in lead colic, is doubtful.

A special variety of icterus, the occurrence of which has been disputed, is urobilin icterus, in which a faint yellow staining of the skin and tissues is produced by urobilin, an oxidation product of bilirubin. This variety includes icterus occurring in pneumonia with hemorrhagic infarctions, when hæmoglobin is extravasated and changed into urobilin. Some investigators look upon urobilin icterus as a slight grade of biliary icterus.

Diseases of the Pancreas.

In the pancreas hemorrhages are now and then observed; they result either from a general hemorrhagic diathesis or else occur idiopathically and spontaneously with partial obstruction of the gland; rarely the hemorrhages are of traumatic or inflammatory origin. Hemorrhages occur also secondarily in connection with necrosis of isolated segments of the gland and of the fatty tissue; in the latter case there may be a formation of hæmatomata and hemorrhagic cysts, followed by the effusion of blood into the abdominal cavity.

Inflammations of the pancreas are rare; occasionally we find an acute purulent pancreatitis. This is either limited to a portion of the gland or the whole of the latter is infiltrated with pus; multiple abscesses may develop from the inflammation, also necrosis of some portions and secondary peritonitis. Diffuse purulent inflammation springs sometimes from the efferent ducts (centrifugally) by the immigration of pathogenic germs from the intestine; besides, a metastatic hæmatogenous inflammation is possible.

Hemorrhagic pancreatitis may develop from primary hemorrhages or from a parenchymatous inflammation of the gland tissue.

Secondarily various forms of pancreatitis result from the extension of the inflammatory process from neighboring organs, *e.g.*, in carcinoma of the duodenum or stomach.

In chronic interstitial pancreatitis the gland appears enlarged and firm, in later stages contracted and board-like in consistence; as in hepatic cirrhosis we find the framework increased and the glandular substance atrophic. Hypertrophic induration develops sometimes in connection with chronic congestion. A special form of atrophic induration results from occlusion of the efferent channel.

Among the neoplasms of the pancreas the first place belongs to carcinoma, which occurs primarily and secondarily. Secondary carcinosis frequently results from direct extension of a primary cancer of the duodenum. The cancer is often confined to the head of the gland. In the majority of cases stenosis of the ductus choledochus and icterus follow.

Sarcoma is very rare, and more frequently secondary than primary.

Tuberculosis of the pancreas is also rare; it occurs either as miliary tuberculosis in general miliary tuberculosis or now and then by extension from the surrounding structures, particularly from tuberculous retroperitoneal lymphatic glands.

Cysts of the pancreas likewise are rare; they are usually retention cysts (ranula pancreatica), or else the cysts develop in connection with interstitial pancreatitis.

Atrophy of the pancreas is observed occasionally in old and marasmic subjects, more frequently in diabetes mellitus. In inflammatory atrophy the gland is flaccid, somewhat darkened, and the lobules are diminished in size and traversed by connective-only to a slight extent from the protoplasm of the liver cells (fatty degeneration); the greater portion is carried to the liver from other organs which have undergone a similar degeneration (fatty infiltration).

In acute yellow atrophy, a process which for the time is still obscure, a considerable portion of the hepatic parenchyma perishes by fatty necrosis; the liver diminishes markedly in size within two to three weeks and appears flabby; on the cut surface red (alcoholic) and yellow (icteric) portions alternate (Plate 54).

2. The second principal form of fatty liver (fatty infiltration, Plate 52, Fig. *a*) is characterized by the fact that the fat is deposited in larger drops in the otherwise intact, merely enlarged liver cells; whether by reason of increased supply, or of diminished combustion, or of both combined. Belonging in part still among the normal conditions is the fatty liver of overfeeding which is met with in obesity; in the higher and extreme degrees (the contained fat rising to forty-four per cent of the fresh substance) it is pathological, as for instance in the artificially produced fatty liver in overfed geese. Another form of this fatty infiltrated liver is frequently found in drunkards, sometimes associated with slight or marked degrees of cirrhosis (fatty cirrhosis).

3. Finally, a pronounced fatty infiltration of the liver is sometimes met with in very cachectic an emaciated persons (cachectic fatty liver), especiall in cases of phthisis, carcinoma, and chronic fevers The origin of this anomaly is rather obscure; pe haps the associated anæmia, increased decompositio of albumin, and consequent decreased combustion